EMSE Egyptian Medical Society of Echocardiography edition

Sedafy's Echocardiography

Step by Step practical approach

FIRST EDITION

Edited by

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لا يسمح بنسخ او تصوير او تسجيل او تداول اى جزء من مواد هذا الكتاب بأى وسيله الكترونية او تقنيه او بأى وسيله اخرى. كما يحظر حظراً تاماً استعمال مواد هذا الكتاب إستعمالاً تجارياً من اى نوع كان إلا بإذن خاص خطياً من الناشر لذالك

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Dedication

Writing the first Egyptian echocardiography book had been a great challenge, this challenge is only accomplished by the presence of some very special and unique people in my life, who never let me down and offered me enormous help support and encouragement.

This work is dedicated to

My father "professor doctor Mamdouh Elsedafy", though not with us now but shared in writing every letter, word and sentence in this book, may his soul rest in peace.

My mother who never lost hope on me and made me the man I am.

My lovely adorable wife for surviving all the difficult times and hours and to my children without whom I could have finished this work one year earlier! They just mean the life to me.

My sisters for always believing in me and encouraging me.

To all my big family for the support they gave me that cannot be described by words.

To all my professors and colleague doctors namely those who directly affected my career

by order of seniority

Professor doctor: Hanaa El saeed

Professor doctor: Mohamed Osama

Professor doctor: Hany Negm

Professor Doctor: Hatem Kholief

Professor Doctor: Mohamed Heikal

Professor Doctor: Ayman Ali Elbadawy

Doctor: Hani Mahmoud

Doctor: Mohamed Abd Elrahman

Doctor: Ahmad Sabry

And my best friend Eslam Mostafa

And finally I would really thank all my trainees who trusted me to teach them echocardiography, by all means you refined my knowledge and upgraded my skills, I am proud to be your mentor.

Introduction.

The no how in learning echocardiography is the perfect understanding of Cardiac Anatomy as well as good knowledge of the physics of U/S Beam? Once these two key points are well known ,echocardiographic views will be so logical not only to understand but also to interpret.

In the EMSE, our new approach in echocardiography training and explanation gained our trainees huge experience in relatively very short time.

We also simplified the concepts of physics into the common day to day nomenclature so that it will be easily understood by Doctors who did not attend a premed collage or had little basic knowledge about physics.

This book is written in a new and a unique way, where all the cardiac pathologies are discussed and explained by the same sequence of steps the echocardiographer will take to finish the echocardiography study.

To join the practical EMSE training semesters contact +2 27929917 /

+2 0111 731 5023 / +02 0106 066 8346 / +2 0122 1990 442, or visit www.sonopro.org or www.echo-training.com

- **The Egyptian Medical Society of Echocardiography "EMSE" awards accredited hours from the American Society of Echocardiography. http://www.aseuniversity.org/ase/events**
- **The EMSE is recommended by the Cardiovascular Credential International "CCI" http://www.cci-online.org/content/review-programs.**

This book handles the various cardiac pathologies only from the echocardiographic point of view together with any relevant clinical associated condition that has an echocardiographic finding.

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Abbreviations

Ao : aorta
Ap 2 ch view : apical 2 chamber view.
Ap 3 chamber view : apical 3 chamber view.
Ap 4 ch view: apical 4 chamber view
AR: aortic regurgitation
AS: aortic stenosis
ASD: atrial septal defect
AV : aortic valve
F : frequency
HCM : hypertrophic cardiomyopathy
IAS: inter atrial septum
IAS: inter atrial septum
IVS : inter ventricular septum
IVS: inter ventricular septum
LA: left atrium
LPA: left pulmonary artery
LV: left ventricle
LVEDD: left ventricular end diastolic dimension
MPG: mean pressure gradient
MR: mitral regurgitation
MS: mitral stenosis
MV : Mitral valve
P: period
PAPVR: partial anomalous pulmonary venous return
PG: pressure gradient
PLAX: parasternal long axis
PPG" peak pressure gradient
PR: pulmonary regurgitation

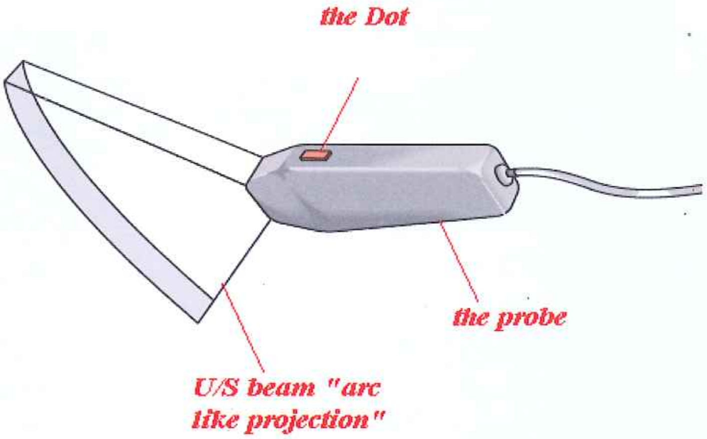
PRF : pulse rate frequency
PRP: pulse repetition period
PS: pulmonary stenosis
PSAX: parasternal short axis
PV : pulmonary valve
RA :right atrium
RPA : right pulmonary artery
RV : right ventricle
SSLA view: supra sternal long axis view
SSSA view: supra sterna short axis view
TR: tricuspid regurgitation
TS: tricuspid stenosis
TV : tricuspid valve
VSD: ventricular septal defect
VTI: velocity time integral.

Chapter 1 :

Echo anatomy : "Echocardiographic views"

The main aim of this chapter is to enable the echocardiographer to optimize a typical view in order to measure accurate parameters. The echocardiographer must correlate the various echocardiographic views to cardiac anatomy.

The probe transmits ultrasound beam in an arc like projection.

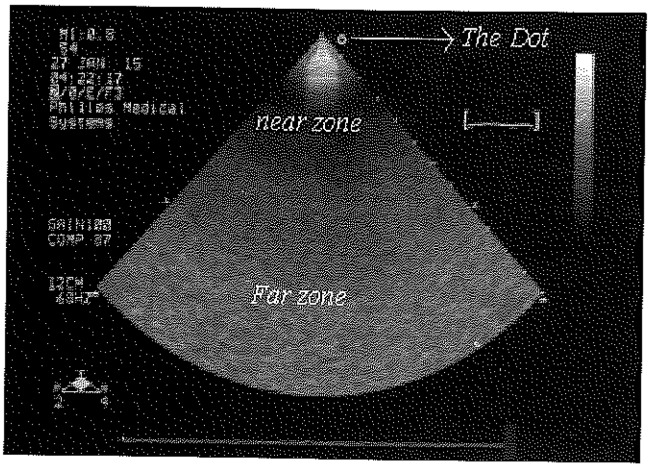


On one side of the probe there is a mark called the "dot" or "Knob".

3 simple concepts to remember in order to understand the echo views are :

1) The probe should be considered as knife handle and the U/S beam is its blade. This blade will cut ***SLICES*** in the heart.
2) The concept of axial image orientation "up & down orientation": The first structure the U/S beam will encounter will be the first structure to be displayed on the screen (near zone). The last structure encountered by the U/S beam will be the last structure displayed on the screen (far zone). This is the concept of the range equation that will be discussed later.
3) The concept of lateral image orientation "side to side image orientation" :
   By directing this dot or knob of the probe in the direction of a structure, this structure will be displayed on the same side of the dot shown on the screen.

A diagram showing an empty view displaying the Dot.



N.B: The dot/ knob will have different shapes according to the manufacturer, but in general it is a mark present on one side of the probe and is absent from the other side.

For the dot/ knob of the probe there is an equivalent dot on the screen, the dot on the screen is fixed on the right side of the screen, and it does not move (unless changed intentionally from the soft ware of the machine). Also this Dot of the screen will have different shapes according to the manufacturer, but in general it is a mark on one side of the screen and it is absent from the other side of the screen.

N.B : The near zone is called Fresnel zone, and the far field is called Fraunhofer zone.

3 rules for orintation :

1) In any apical view the first structuer encounterd by the beam is the apex.
2) In any parasternal view the first structure encounterd by the beam is the RV.
3) In any subcostal view showing the heart the first structure encounterd by the beam in the liver then the RV.

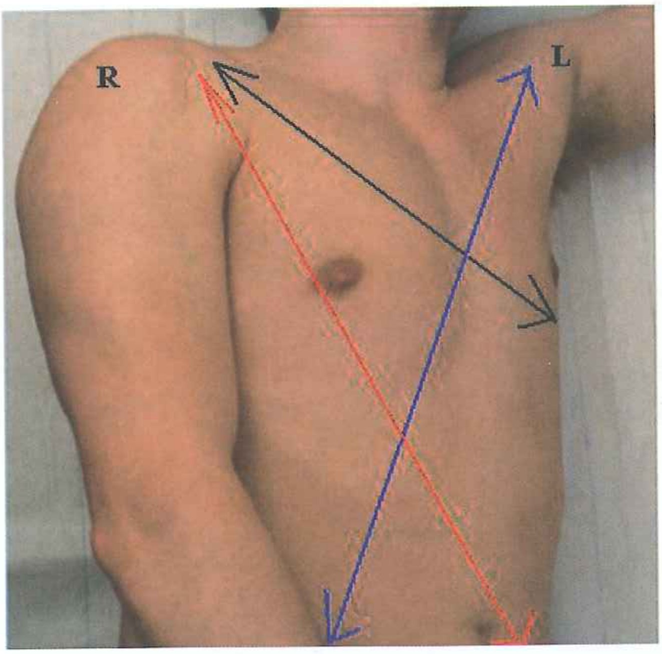
Various motions of the probe :

The 2 basic rules for probe motions :

1) All the motions must be done by the fingers and NEVER by your wrist.
   Clinical tip: It is accepted to aid the motions by your other hand instead of using your wrist to move the probe. (This rule is used to gurantee easy manilpulation of the prob).
2) Almost always support your wirst on the patient.
   Grip the probe using all your five fingers TIPS, 4 fingers behined the probe and the thumb from infront (optional).

N.B : In all apical and parasternal views the dot of the probe will be directed towards the pateint's left shoulder, except in the PLAX and the apical 3 chamber views, where the dot will be directed towards the right shoulder.

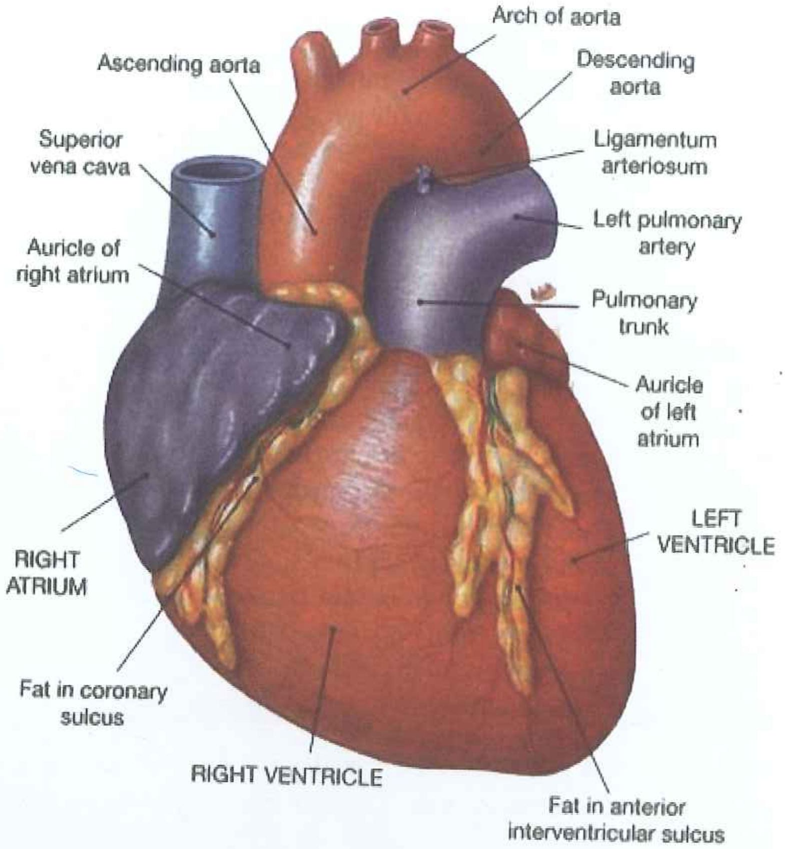
Map 1

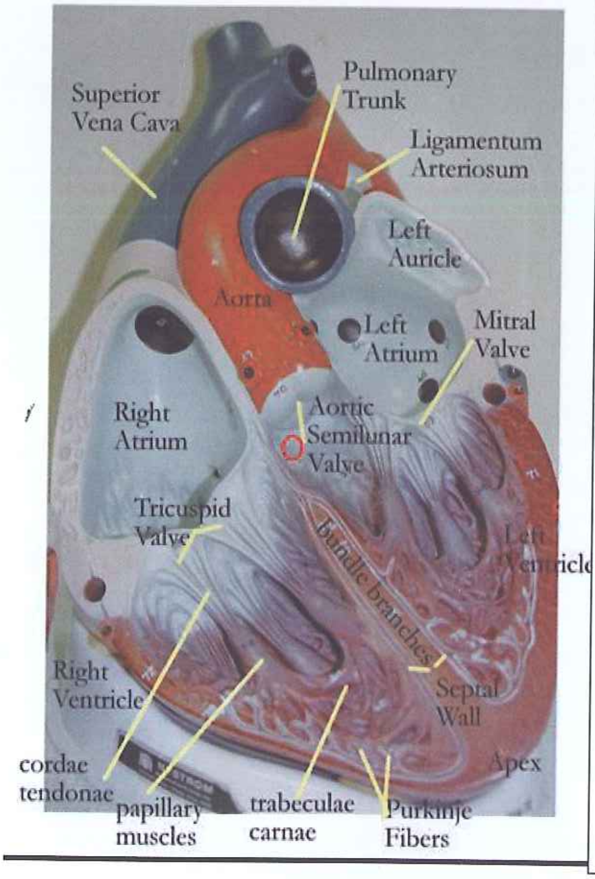


Any motion of the probe that is parallel to a specific axis is called pendular motion (rocking motion). Any motion that makes an angle with a specific axis is called tilting motion.

The key point to understand any echocardiography view is to correlate the echo view to its precise anatomical cut section that is illustrated in the various cardiac models given in the next review.

Basic cardiac anatomy that aids echocardiography understanding.

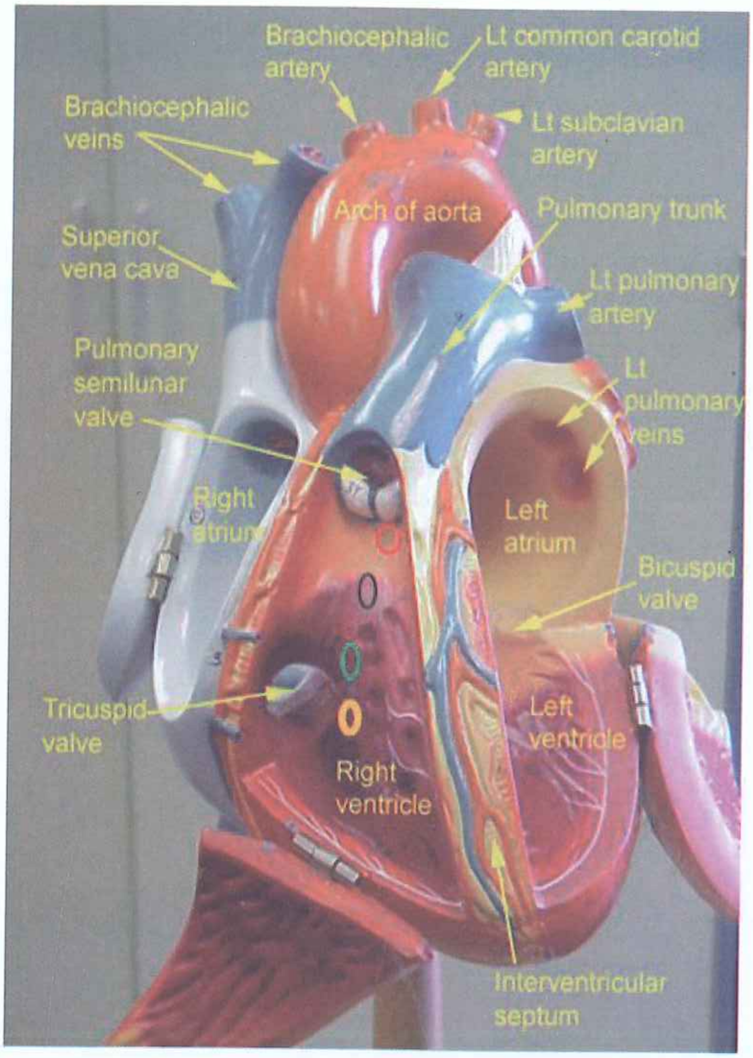




Anatomical cardaic model showing the left side of the heart with the main pulmonary artery and pulmonary valves removed

Notice the position of the aortic valve as being wedged between the mitral and the tricuspid valves. The pulmonary valve should be superior to and in front of the aortic valve and directed towards the RV. See the next cardiac model.

Notice the site of the red circle just below the aortic valve, this is the approximate site of the membranous inter ventricular septum.



An Anatomical model showing the right side of the heart.

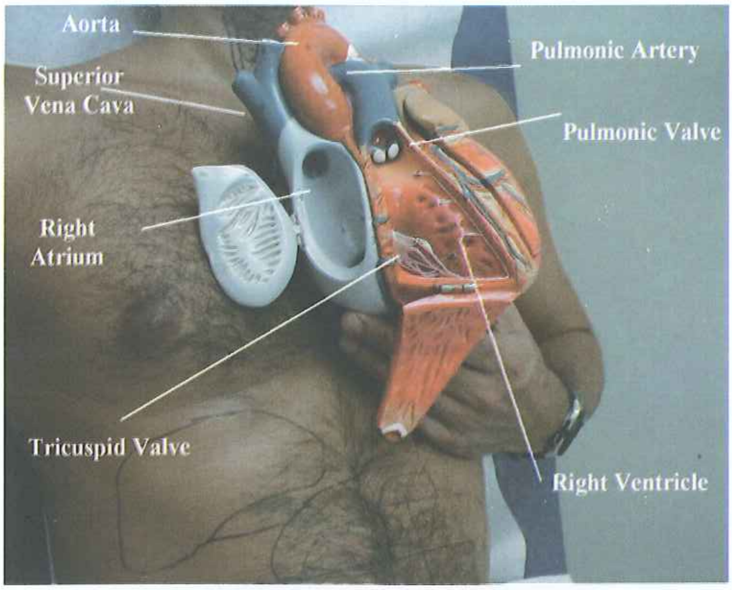
The aortic valve is over masked by the pulmonary valve and pulmonary artery.

Notice the red circle just downwards the pulmonary valve, this is the approximate site of the subpulmonic interventricular septal defect.

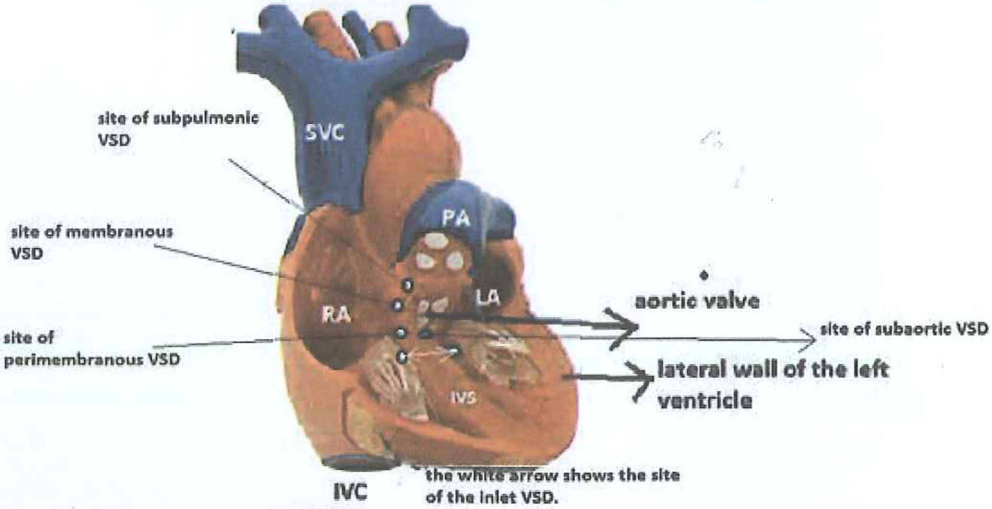
Notice the position of the black circle, this is the position of the membranous VSD.

Notice the position of the green circle near to the tricusped valve, this is the site of the peri-membranous inter ventericular septum.

Notice the site of the orange circle, this is the site of the "inlet" interventricular septum.

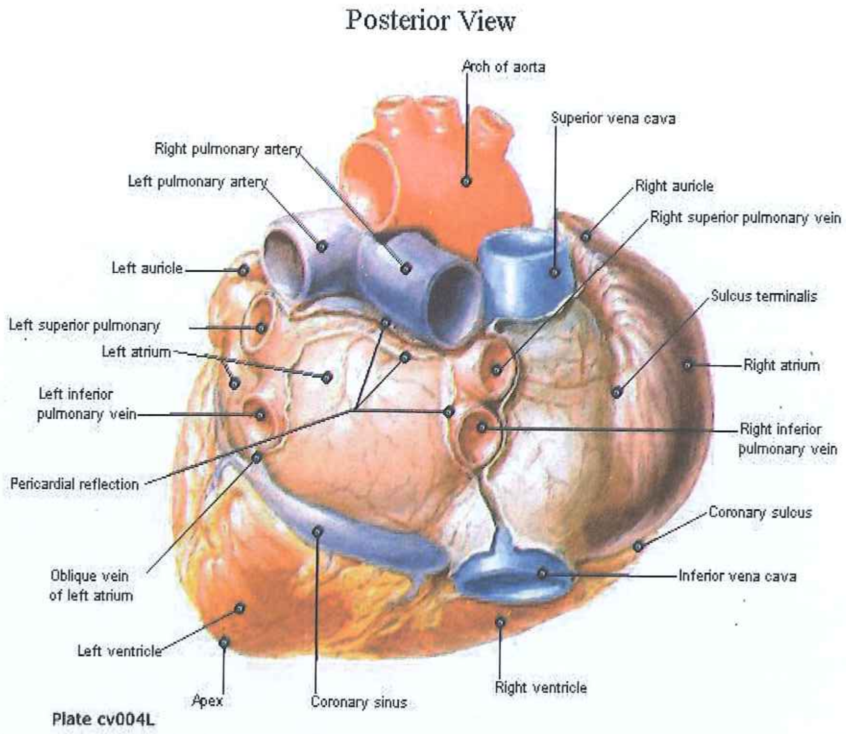


Notice the normal cardial position, notice the normal cardaic base to apex axis.


site of subpulmonic VSD
SVC
site of membranous VSD
PA
RA
LA
aortic valve
site of perimembranous VSD
site of subaortic VSD
lateral wall of the left ventricle
IVS
IVC
the white arrow shows the site of the inlet VSD.

Notice the normal antero-posterior pulmonary-aortic criss cross pattern relationship. The part of the interventricular septum between the mitral and the tricusped valve is called the inlet septum.

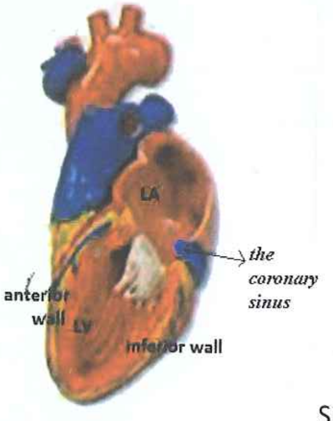
Notice the various sites of the VSD's.



Notice that the most posterior chamber of the heart is the LA, however the most posterior structure of the heart at a higher level is the bifurcation of the pulmonary artery, and the most posterior structure of the heart at a lower level is the left ventricle.

Notice that the descending aorta will continue inbetween both pulmonary branches (the right pulmonary aretry and the left pulmonary artery), then posterior to the left atrium.

Notice the position of the coronary sinus in the posterior atrio-ventericular groove.

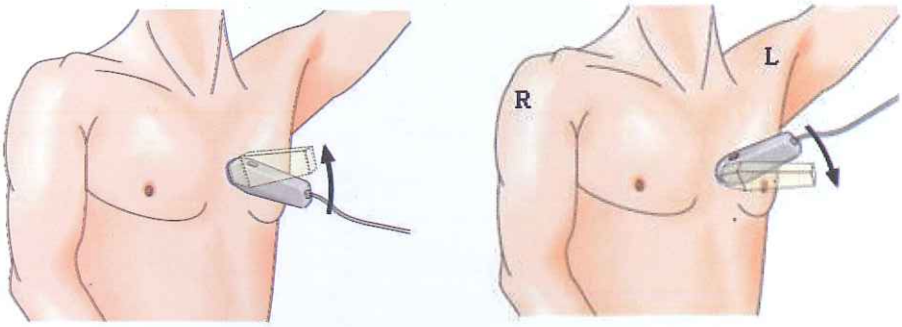


Side view of the heart with lateral wall removed, showing the anterior wall and the inferior wall.

(N.B in examples 1,2 &3 the probe is placed in the parasternal region and the dot is directed towards the right shoulder)

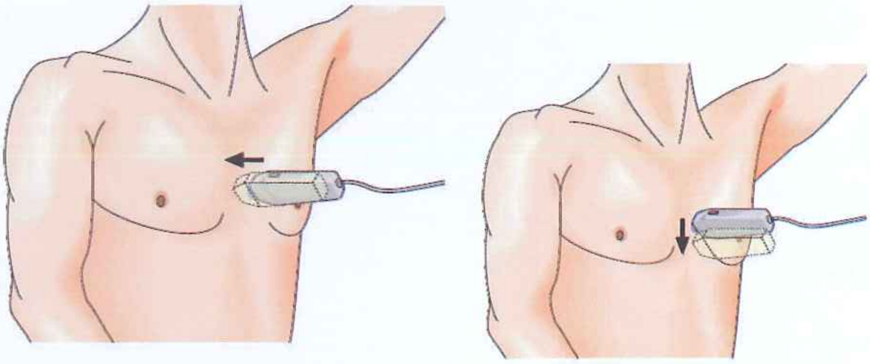
The same concept of motions should be imagined and applied with different probe positions. i.e. when the probe is placed in the apical position.

All motion directions are named by the direction of the U/S beam, i.e. an anterior tilt means that this tilting motion will direct the U/S beam anteriorly (this means that the tail of the probe will be directed to the opposite side).

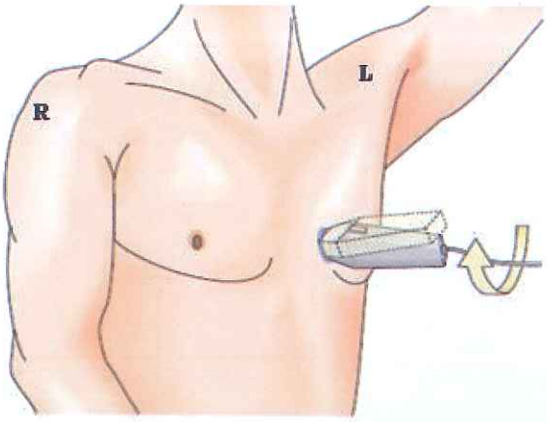


Example 1 :

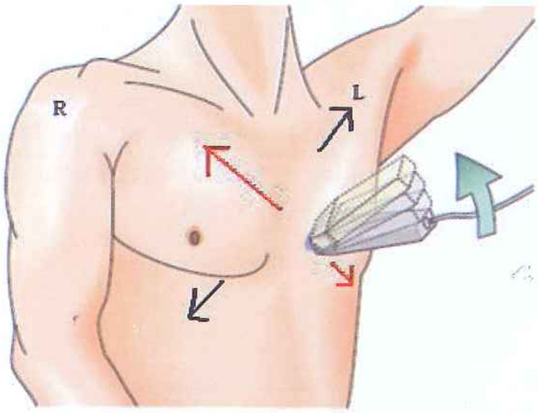
The concept of pendular (rocking motion): a motion parallel to the long axis of the probe (i.e. a motion in the same direction of the dot). In this example the dot is directed towards the right shoulder so the motion is parallel to an axis between the apex and the right shoulder (parallel to the black line in Map 1 in page 3), if thae dot is directed towards the left shoulder, the pendular motion will be parallel to an axis between the left shoulder and the right hip (parallel to the blue line in Map 1).



Example 2 : The concept of sliding motion. i.e. shifting the probe medial/ lateral or upwards/ downwards.

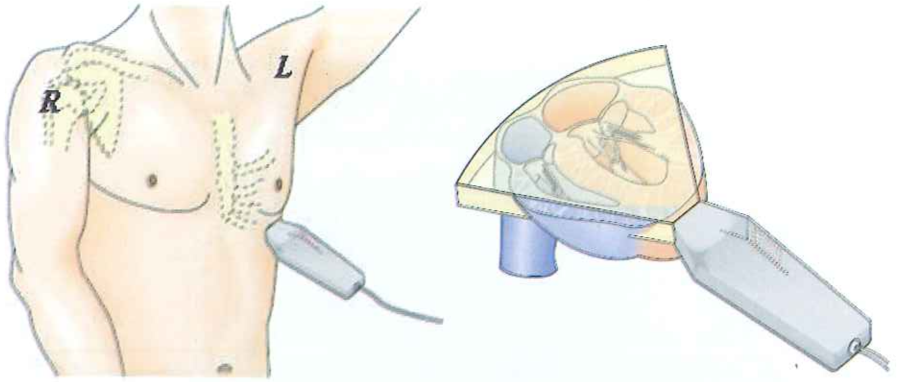


Example 3 : The concept of the rotation motion of the probe. By rotating the probe either clock wise or anticlock wise BUT with the same axis. i.e. without any tilting, sliding or rocking motions. The rotation motion will direct the dot towards the right shoulder or the left shoulder.



The concept of tilting motion of the probe. A motion perpendicular to the long axis of the probe. In this example the dot is directed towards the left shoulder, so the tilting motion will either direct the U/S beam crainially and towards the right shoulder *(so the tail of the probe will be directed caudally towards the left hip) see the red arrows*, or the tilting motion will direct the U/S beam in the opposite direction caudally and towards the left hip (so the tail of the probe will be directed crainially towards the patient's right shoulder) see the red arrows too.

If the dot of the probe is directed towards the right shoulder, the tilting motion will either direct the U/S beam crainially towards the left shoulder *(so the tail of the probe will be directed caudally towards the right hip) see the black arrows*, or the tilting motion will direct the U/S beam caudally towards the right hip (so the tail of the probe will be tilted crainially towards the left shoulder) see the black arrows too.

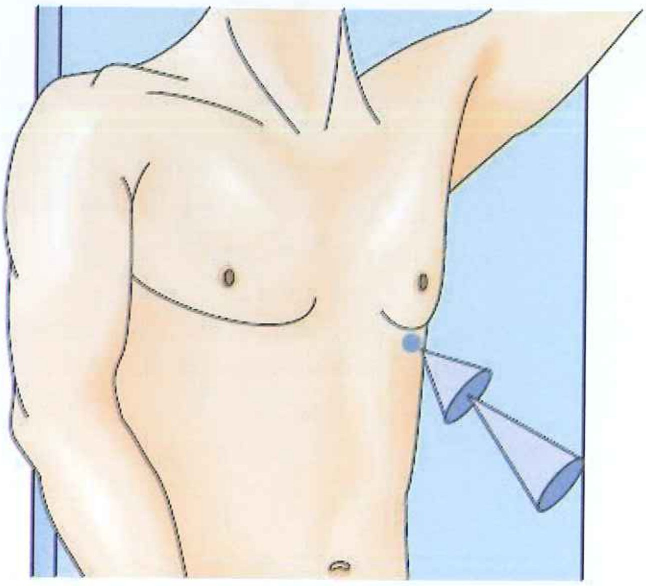


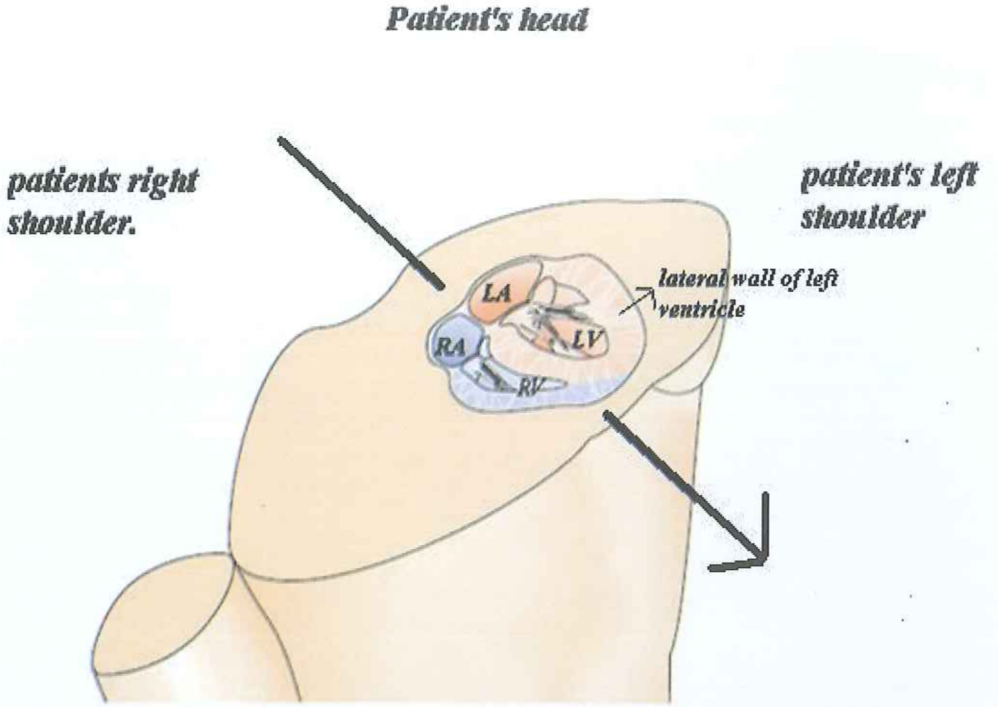
In this example the probe is placed over the apex and the dot is directed towards the left shoulder, the tillting motion in this position will either direct the U/S beam anteriorly (so the tail of the probe will be tilted caudally), or the tilting motion will direct the U/S beam posteriorly (so the tail of the probe will be tilted anteriorly).

The pendular motion in this position will be parallel to an imaginary line between the left shoulder and the right hip (parallel to the blue line in Map 1).

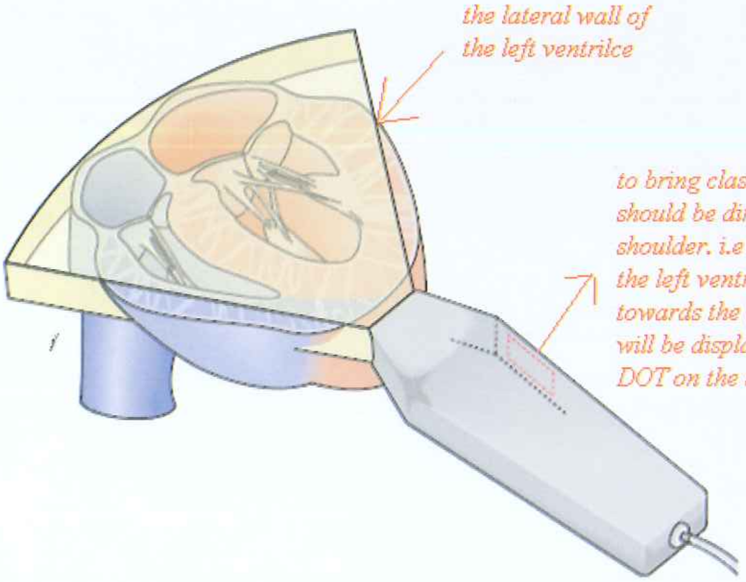
Apical 4 chmaber view "apical 4ch view" :

To bring an apical 4 chamber view the probe must be placed over the apex, and by understanding the base to apex axis of the heart (as being normally levocardia) the beam should be directed up and in a medial orintation towards the base of the heart. i.e. the U/S beam should be parallel to the blue line in Map 1 page 3.





real anatomical orintation for an apical 4ch view cut section, with the probe placed at the apex the U/S beam will first encounter the apex, so the apex will be firstly diplayed at the top of the screen, notice the normal base to apex axis of the heart : levocardia as shown by the arrow

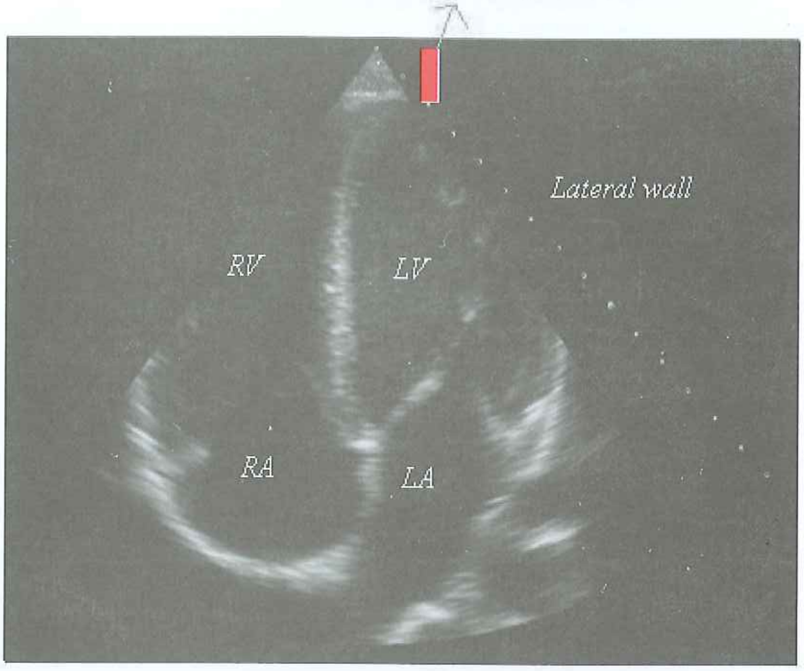


the lateral wall of the left ventrilce

to bring classic apical 4ch view the knobe should be directed towards the left shoulder. i.e towards the lateral wall of the left ventricle, by directing the knob towards the lateral wall, the lateral wall will be displayed at the same side of the DOT on the screen

Slice number 1, N.B this slice is in a more posterior plane to slice number 2. (see slice number 2 later)

the Dot is classically displayed at the right side of the screen



By applying the 3 concepts discussed before, the result will be:

The apex is displayed at the top of the screen "at the near zone" (apply concept number 2)

The lateral wall will be at the same side of the Dot of the screen (apply concept number 3)

The cardiac walls are the posterior septum and the lateral wall (apical, mid and basal parts are shown from above downwards).

Definition of the typical apical 4 chamber view:

1) Vertical view. The curser line (white dotted line) can be aligned in a parallel manner to the blood as it flows from the LA to the LV.(as shown)
2) Showing the true apex. The true apex is the thinnest part of the cardiac wall (i.e. thinner than the septum and the lateral wall)

The EMSE Stepwise approach to adjust the apical 4ch view :

The key point to optimize an apical 4ch view is to direct the U/S beam exactly parallel to the arrow shown in diagram "A".

Place the probe over the apex. Direct the Dot of the probe towards the left shoulder of the patient and direct the beam medially towards the base of the heart as shown in diagram "A". A common mistake is that the beginner echocardiographer does not direct the beam towards the base of the heart. For example some trainees place the probe over the apex but direct the beam towards the patient's abdomen instead of the cardiac base.

Slide the probe up and down with the U/S beam always directed towards the base of the heart.

If the heart is not localized repeat the same sliding motions "up & down" in a more medial or a more lateral place. Keep in mind that the beam should always be oriented up and medially towards the base of the heart.

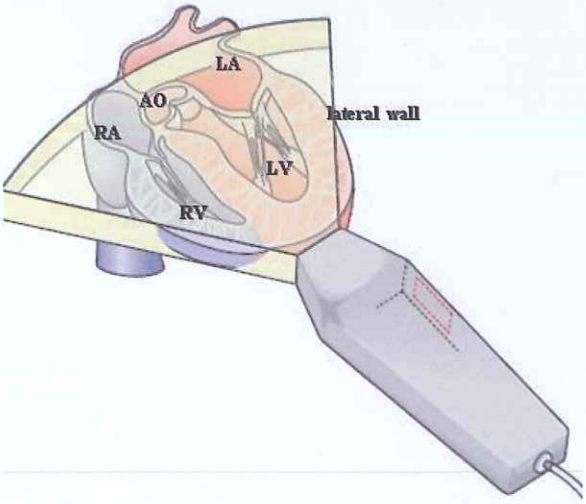
After localizing the heart, three main problems may be encountered :

1) If the view is not centralized → fine pendular motion "rocking motion" will centralize the view. (Extreme caution should be taken not to add a sliding motion with the pendular motion.)
2) If the left atrium (LA) is not optimized→ fine tilting motion will open the LA.
3) If the right ventricle (RV) is not optimized → fine rotation motion will open the RV.
4) In abnormal cardiac axis or hugely dilated dimensions, the Ap4 ch view may be optimized by rotating the dot in a more clockwise pattern, i.e. the dot will not be directed towards the left shoulder, but it will be directed more transversely. The key point is that the echocardiographer has to rotate the probe until all the 4 chambers are opened.
5) Take care that in dilated LV the apex may be shifted downwards, downwards and laterally, or even in the mid axillary line or beyond. i.e. search for the apex !

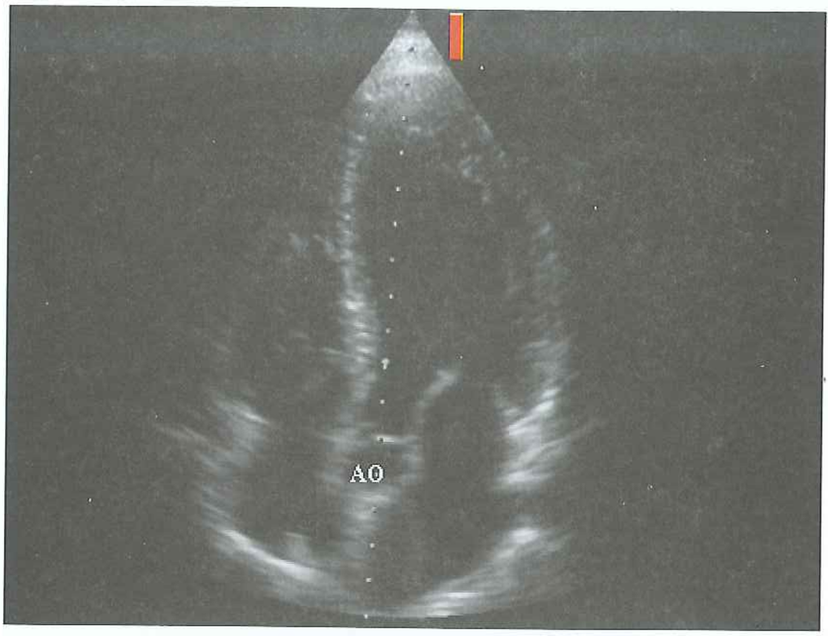
Apical 5 chamber view "Ap 5ch view"

From an optimized Ap 4 ch view, tilt the probe anteriorly (i.e. tilt the tail of the probe posteriorly) , this will direct U/S beam in a more anterior plan where the aorta is placed and wedged between the mitral valve and the tricuspid valve.

The view will look much like the Ap4 ch view but showing the aortic valve in its anatomical position between the mitral and the tricuspid valves.



Slice number 2 . N.B this slice is in a more anterior plan to slice number 1.



Apical 5 ch view. (the cardiac walls are the lateral wall and the anterior IVS)

Definition of the typical apical 5ch view :

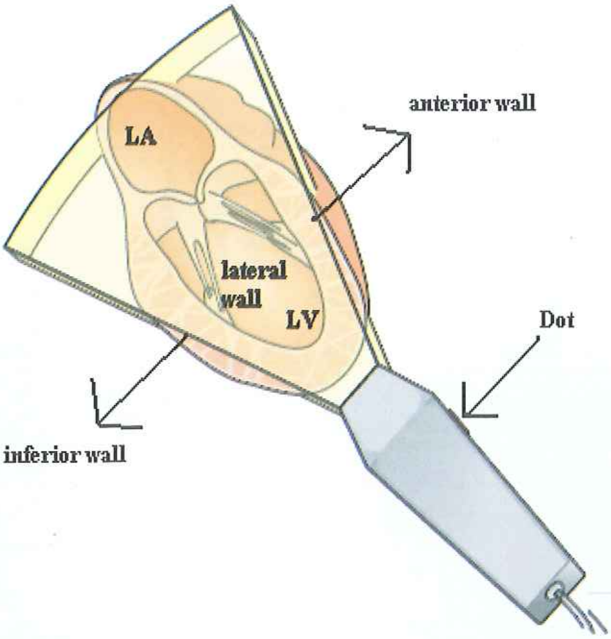
1) Vertical view.
2) Showing clear LVOT and aortic valve in a way that permits the curser to be aligned parallel to the blood flow from the LV to the aorta.

The **EMSE** stepwise approach to adjust the Ap 5 ch view is the same as in Ap 4ch view.

Apical 2 chamber view "Ap2 ch view" :

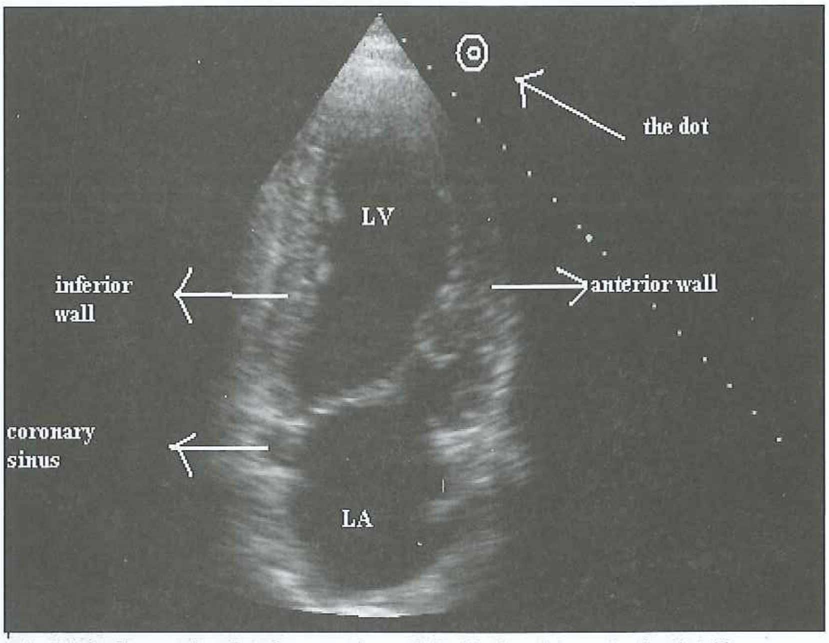
From an optimized apical 4 ch view rotate the probe anti clock wise rotation so that the dot is directed towards the left mid clavicular line. The echocardiographer must keep the axis unchanged during this rotational motion. If this is the situation, the view will simply change from an apical 4 ch view to an apical 2 ch view. If the echocardiographer changed the axis i.e. he added any other motion to the rotation, the view will be lost.

By directing the dot anteriorly towards the left midclavicular line the dot will be actually directed towards the anterior wall of the heart, so the anterior wall will be displayed towards the dot on the screen (see concept number 3).



apical 2ch view, N.B : the lateral wall will not be displayed in the echo view because it will be lateral to the U/S beam. i.e. the U/S beam will not cut over the lateral wall, BUT the U/S beam will cut over the anterior wall and the inferior wall. notice that the dot of the prob is directed towards the anterior wall, so the anterior wall will be displayed towards the dot on the screen.

Slice number 3 , N.B this slice is vertical to slice number 2.



apical 2ch view, notice that the anterior wall is displayed towards the dot, (because the dot of the probe was rotated anteriorly "anti clock wise" towards the anterior wall

EMSE step wise approach to adjust the apical 2ch view:

From an optimized Ap 4 ch view rotate the probe in an anticlockwise manner until the RV is totally closed (if the patient is within normal cardiac axis and without severe chamber dilatation the dot should be pointed towards the left mid clavicle, otherwise the echocardiographer has to rotate the probe until the RV is closed)

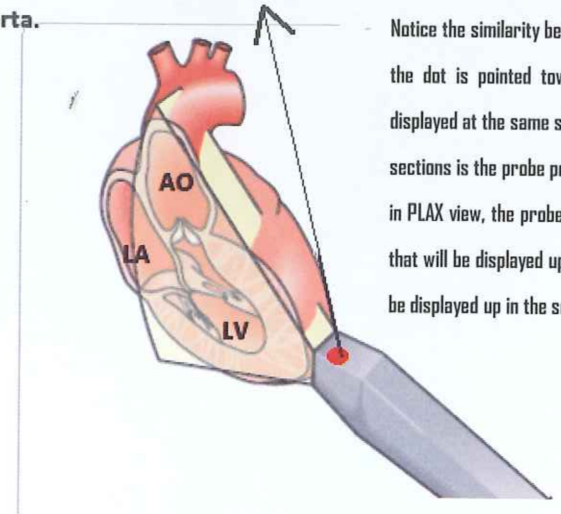
The main pitfall during this rotational motion is that a beginner echocardiographer may add a tilting motion. If this happens, the LA will be closed. To open the LA, apply fine tilting motions in both directions until the LA is shown.(based on our experience in echocardiography training, the beginner echocardiographer will tilt medially by mistake, so to correct this wrong motion, usually a lateral tilt will open the LA)

The second pitfall is the "added on" sliding motion. If this is the situation, the view will be totally missed. So, try to relocate the cardiac apex by fine sliding motions. (based on our experience in echocardiography training the beginner echocardiographer usually slide medially by mistake, so to correct this wrong medial sliding try to slide lateral)

Apical 3 chamber view:

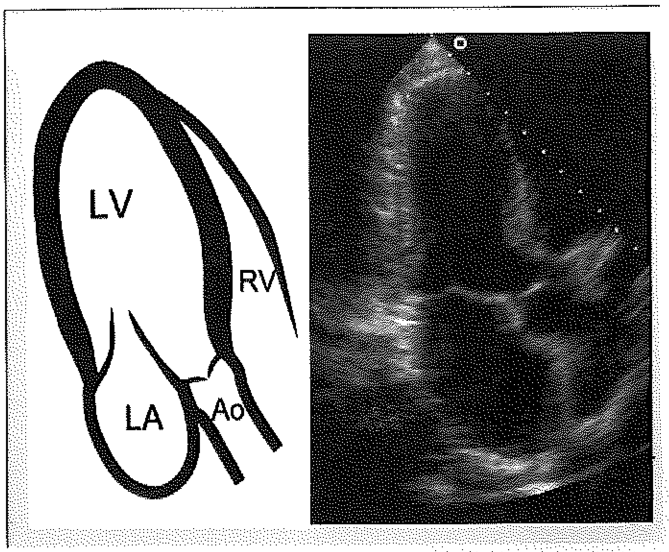
From an optimized Ap 2 ch view, rotate the probe in an anti clock wise manner until the aorta is displayed on the right side of the screen. (in normal individuals the rotation will be typically 60 degree from the apical 2 ch view position). Of notice is that the Ap 3 ch view orientation is the same as the parasternal long axis view "PLAX view" orientation but from an apical position , i.e. the dot must look towards the right shoulder (as in PLAX view) .

Based on our training experience this is the most difficult view for a beginner echocardiographer to aquire. This is due to the two rotations that should be applied from the apical 4 chamber view (Ap 4 ch view→60-90 rotation → Ap 2 ch view →60 degree rotation → Ap 3 ch view). By the end of such rotations the probe grip is usually distorted and mal handed by the beginners, so remember that all the motions performed must be done by your fingers and NEVER by your wrist.



the dot is directed towards the aorta.

Notice the similarity between this cut section and the cut section of the PLAX view. In both the dot is pointed towards the ascending aorta, so in both views the aorta will be displayed at the same side of the dot on the screen. The only difference between these cut sections is the probe position; in the Ap 3 ch view, the probe is placed over the apex, while in PLAX view, the probe is placed over the RV. So in the Ap 3 ch view, the first structure that will be displayed up in the screen is the apex. In the PLAX view, the first structure to be displayed up in the screen is the RV.



Apical 3 chamber view, by rotating the probe and directing the dot of the probe towards the right shoulder the dot will be actually directed towards the aortic valve, so the aortic valve will be directed towards the dot in the screen.

Parasternal views :

As the right ventricle is anatomically the most anterior chamber of the heart, so in any parasternal position the right ventricle will be the first structure encountered by the U/S beam and it will be the first structure to appear in the upper part of the screen.

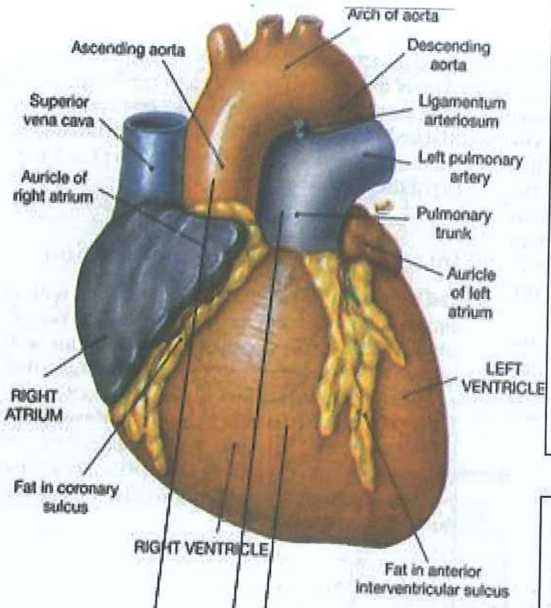
Always remember that the LA is the most posterior chamber of the heart.

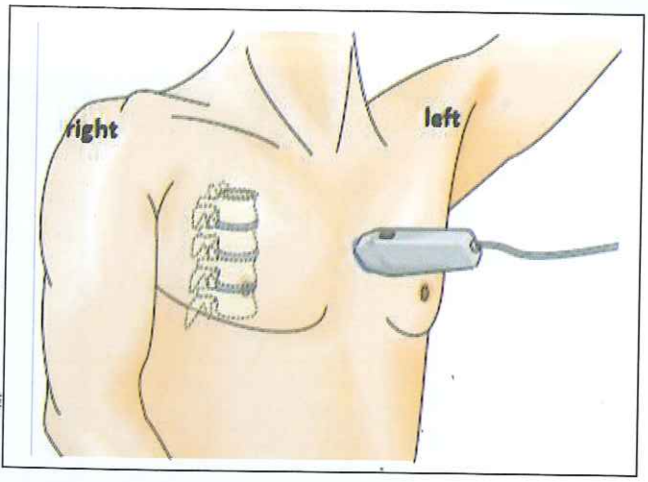
Parasternal long axis view " PLAX" :

To bring a PLAX view the probe should be placed in a way that U/S beam cuts parallel to the long axis of the heart, i.e. the beam should be parallel to the black line in Map 1. The long axis of the heart is an imaginary line joining the cardiac apex and the right shoulder (the black line in map 1)

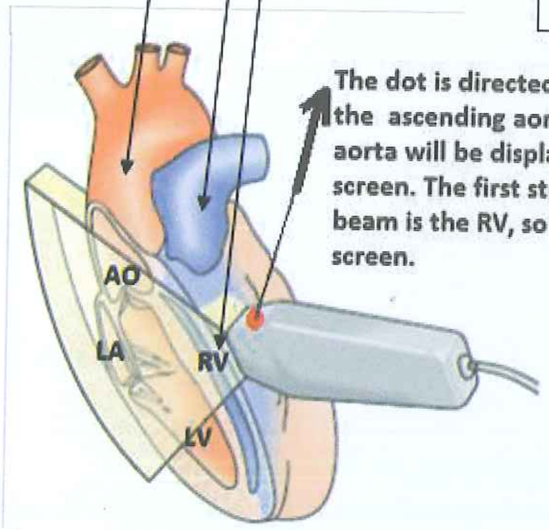
By placing the probe in this way the U/S beam will cut as shown in the echo slice shown below.

To bring a classic PLAX view the dot of the probe will be directed towards the right shoulder, by directing the dot that way, the dot is actually directed towards the base of the heart. So the base of the heart will be displayed at the same side of the dot shown on the screen.



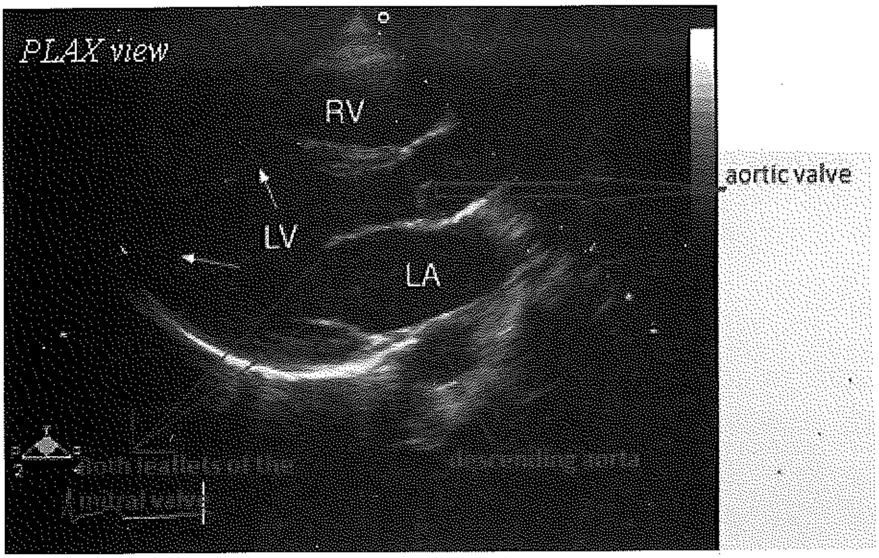


Notice that part of the LV lies anatomically behind the RV, this relationship is shown in the slide below.



The dot is directed towards the base of the heart & the ascending aorta, so the base of the heart & the aorta will be displayed towards the dot in the screen. The first structure encountered by the U/S beam is the RV, so the RV will be displayed up in the screen.

Anatomical cut section in the heart to corrrelate the U/S imaging with the real anatomy



The cardiac walls are the anterior septum and the posterior wall (basal and mid parts are shown), notice the similarity between this view and the Ap 3 ch view, both cut sections are the same but from different orientations.

Definition of the typical PLAX view:

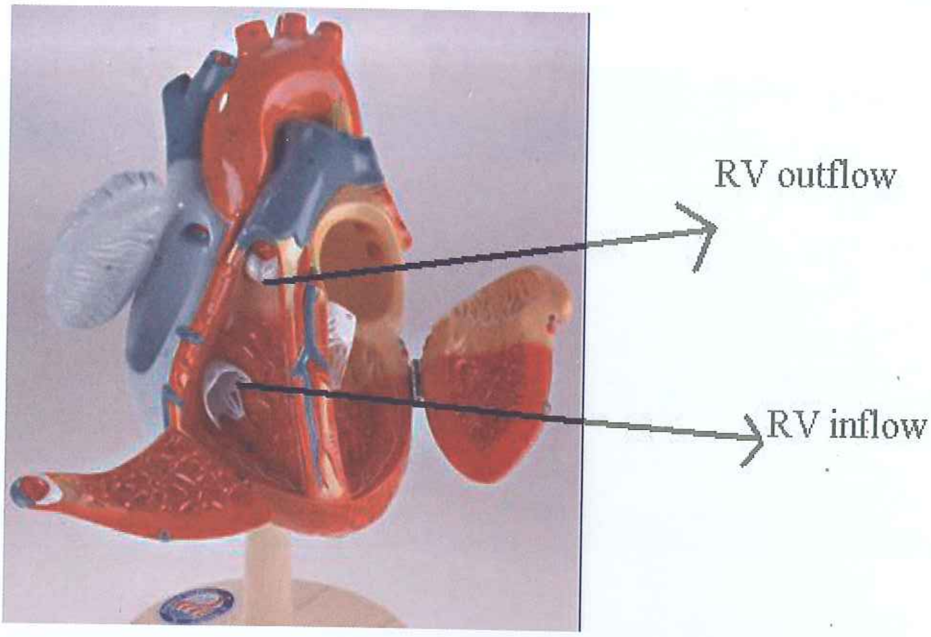
1) Horizontal orientation i.e. permits the curser perpendicular to the IVS.
2) Showing a clear LVOT
3) Not showing the cardiac apex.

EMSE step wise approach to find and adjust the PLAX view:

1) Start by placing the probe in the 2nd intercostal space in the left parasternal region as illustrated before.
2) Slide the probe down wards while keeping the orientation of the dot towards the right shoulder until the heart is imaged.
3) Once you have localized the heart apply tilting motions until the LVOT becomes clear.
4) Use the rotation motion of the probe to eliminate the cardiac apex from the view.
5) Use the pendular motion to centralize the view in the middle of the screen.
   N.B during sliding motion, remember to restrict your probe position in the parasternal area do not slide laterally nor over the sternum.

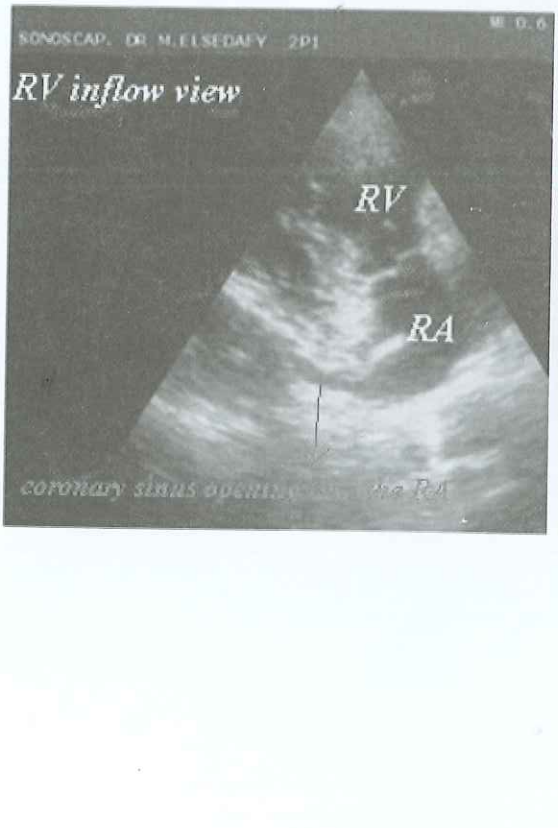
Modified PLAX views:

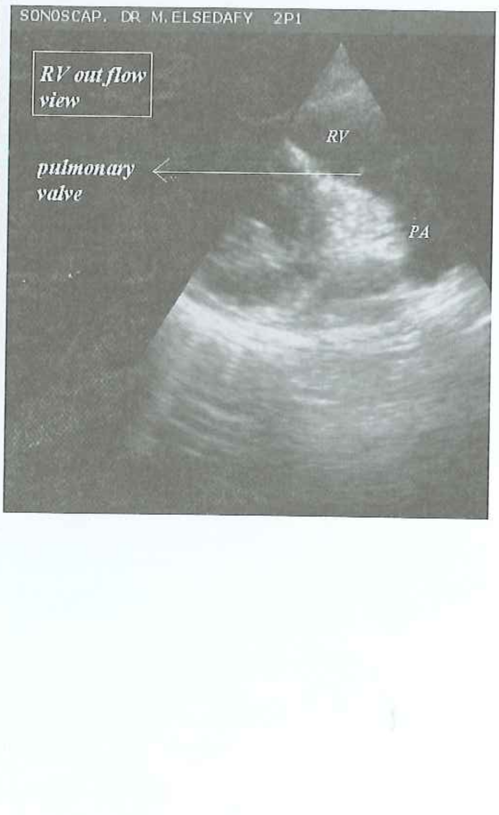
From the PLAX view tilting the probe caudally and cranially will Image the RV inflow view, and RV outflow views respectively.



In the PLAX cut section, the U/S beam should cut through an imaginary line between the pulmonary valve and the tricuspid valve. Tilting this beam caudally will cut through the right ventricle then the TV then the RA. So the RV will be firstly displayed up in the screen then the TV then the RA in the far field.

Titling the U/S beam cranially will also cut firstly through the RV, but due to the cranial tilt the U/S beam will then cut through the PV then the PA, so, the RV will be also the first structure to be displayed up in the screen then the PV then the PA in the far zone.



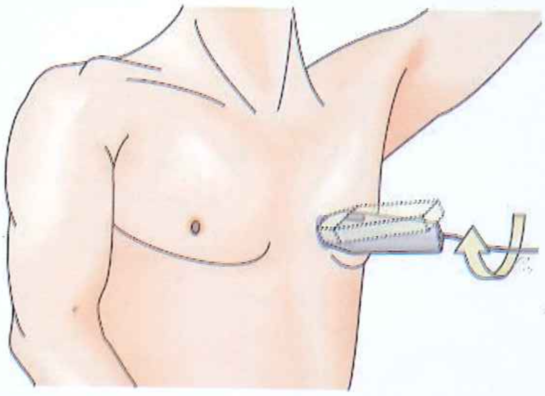


Parasternal short axis probe orientation "PSAX views":

To bring the PSAX orientation, rotate the probe in an anti clock wise manner until the LV becomes circular. In classic situations this will be accomplished by rotating the dot (from the PLAX position) 90 degrees towards the left shoulder. However in abnormal cardiac axis the degrees of rotation will vary and the echocardiographer has to rotate until the view is optimized. ***whatever the PSAX view, the RV will be the first structure encountered by the U/S beam. So, the RV will be displayed in the near zone.***

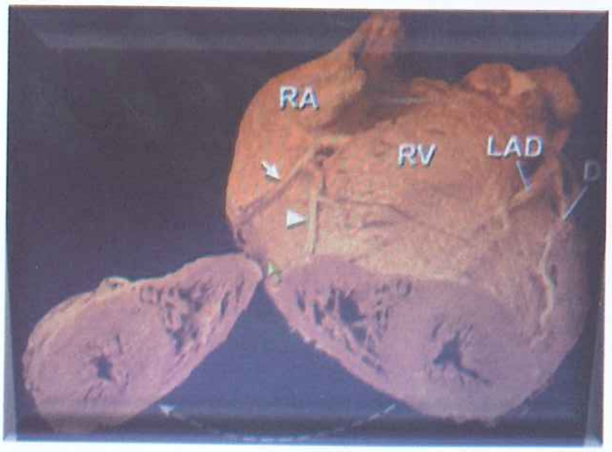
owing to the inevitable fine tilting motions that will occur during the probe rotation and the subtle variation in the cardiac axis & positions, 3 cut sections can be encountered after the rotation, 1) the great vessels level (basal level)., 2)the mitral valve, or 3) the papillary muscles

Now the dot is towards the left shoulder and the U/S beam is perpendicular to the *black* line in Map 1 in page 3 . i.e. the U/S beam is in the left parasternal position and is placed over and parallel to the *blue* line in Map1.

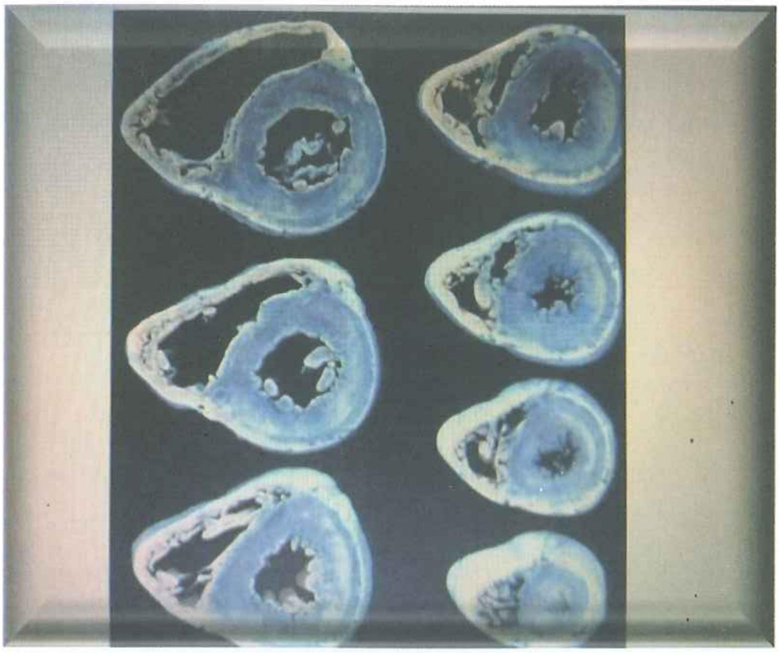


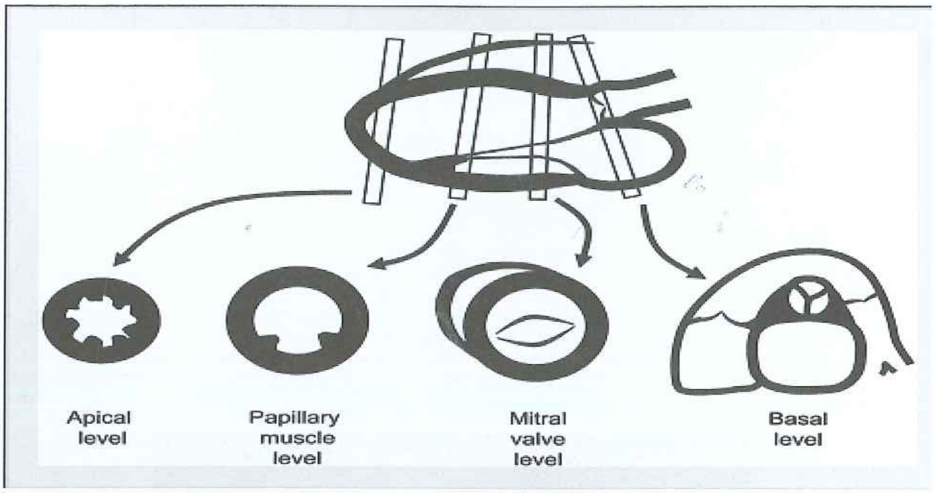
On rotating the transducer 90° clockwise, the heart is imaged in the parasternal short-axis view.

The concept of probe rotation from the PLAX orientation to the PSAX orientation.



By rotating the probe 90 degrees from the PLAX position the heart will be sliced in a short axis manner.

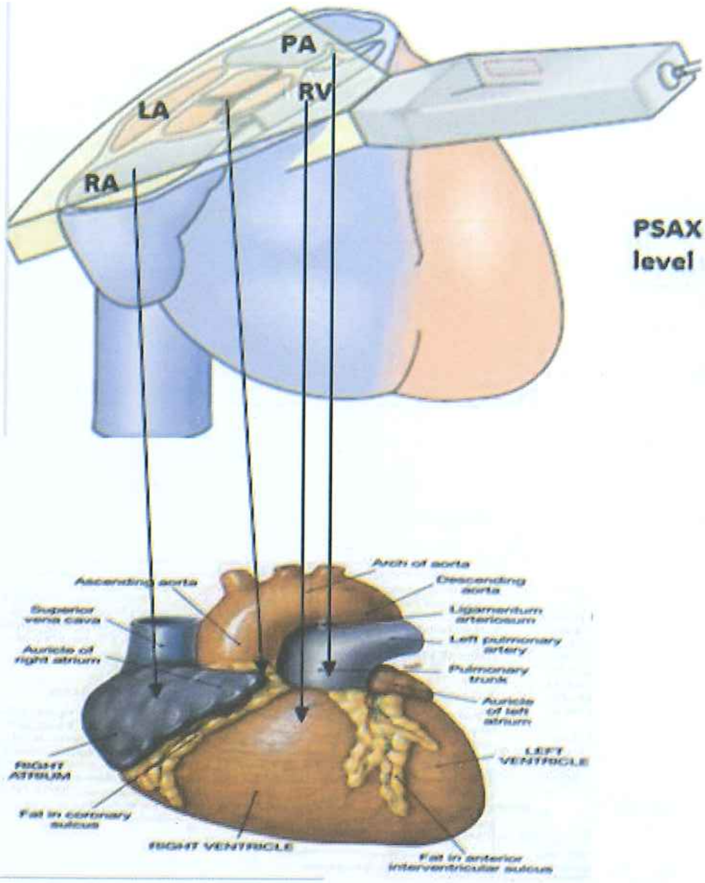




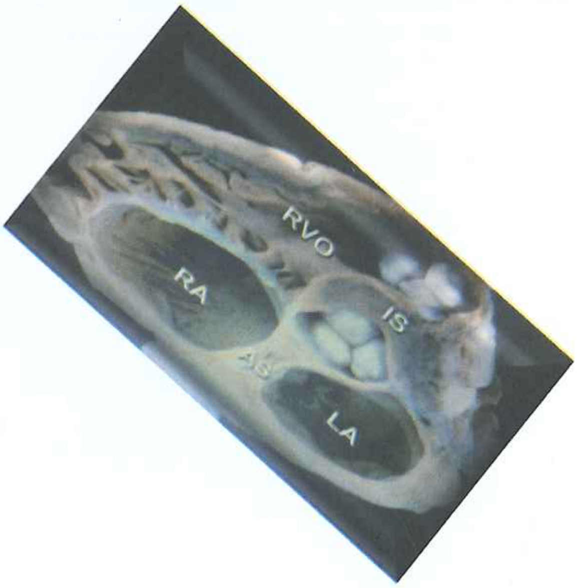
Using tilting probe motions the echocardiographer can slice the heart as shown.

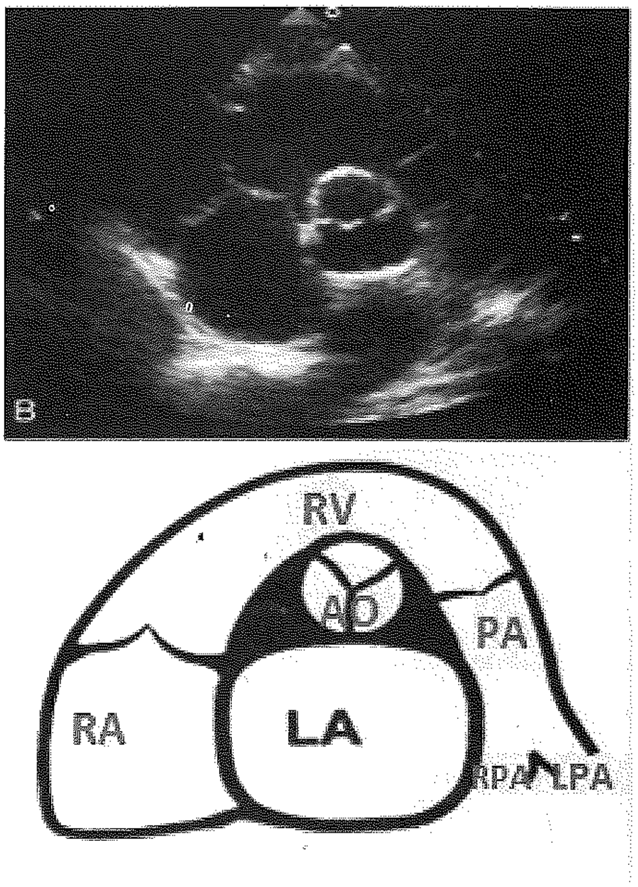
Anatomical section in the heart : notice that the most anterior chamber of the heart is the RV, this means that the first structure the U/S beam will encounter is the RV, so the first structure that will appear up in the screen is the RV, notice also the direction of the dot, by directing the dot towards the left shoulder the dot is acutally directed towards the PA, so the PA will be directed towards the dot in the screen.

1) Great vessels level view "basal level"



PSAX view great vessels level

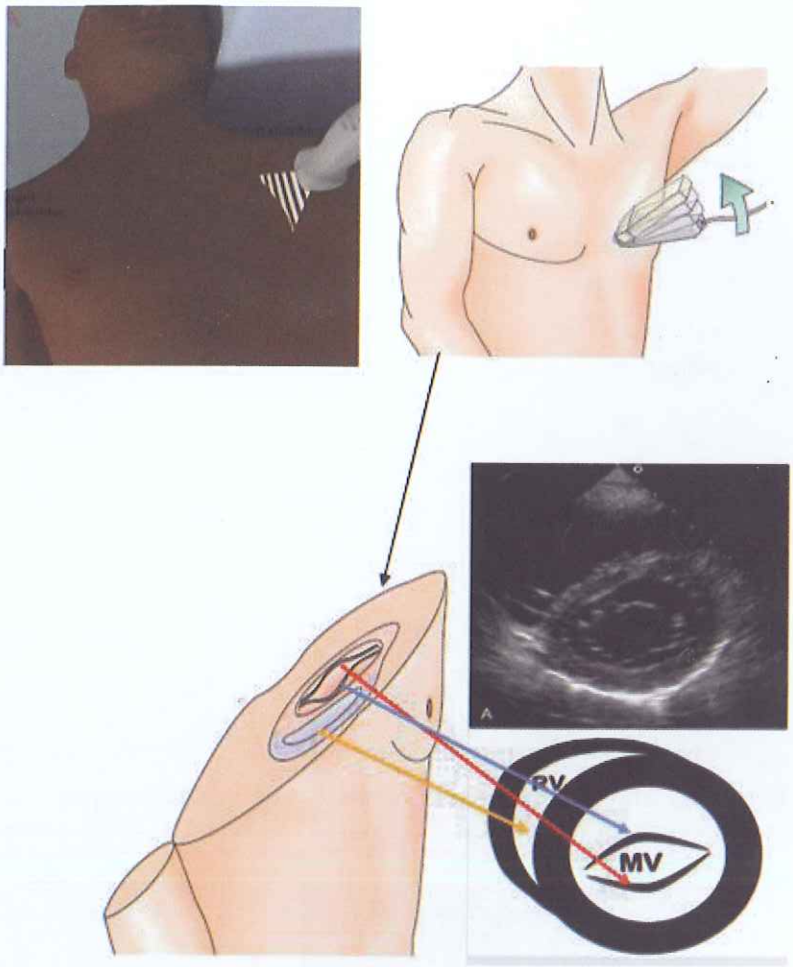




PSAX basal level "great vessels level. Notice that the left atrium is anatomically the most posterior structure of the heart, and the RV is the most anterior structure of the heart,

2) PSAX mitral valve level view:

By further caudal tilting, the dot is still directed to the left shoulder but the cut section is more caudal (at the mitral valve), and the dot is actually directed towards the LV (lateral wall of the LV) so the LV will be displayed towards the dot as shown below. The U/S beam "still" firstly encounters the RV so the RV is displayed up in the screen (in the near zone).



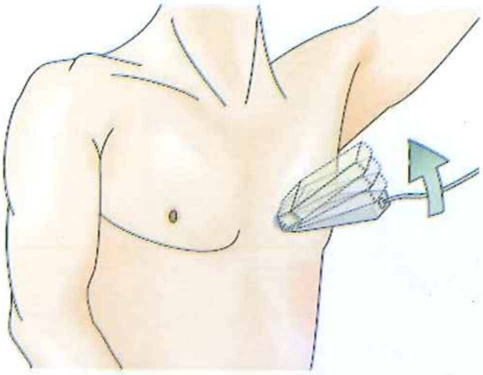
The concept of the probe position in the PSAX view, the dot has to be directed towards the left shoulder.

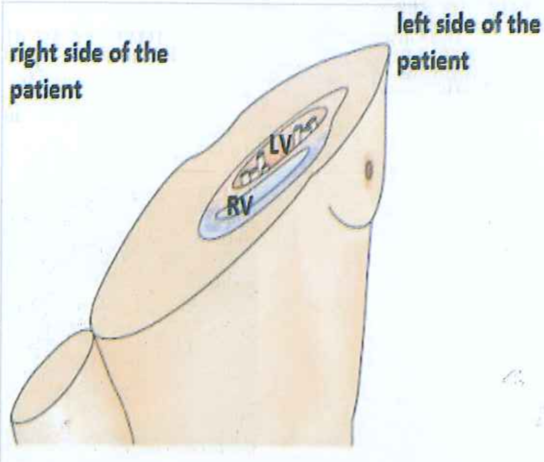
The red arrow is pointing to the posterior mitral leaflet.

The blue arrow is pointing to the anterior mitral leaflet.

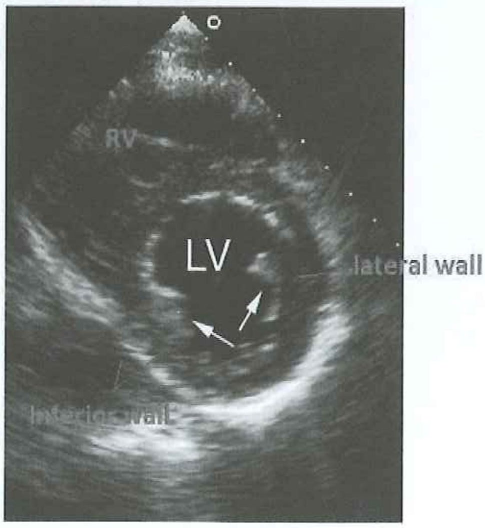
The orange arrow is pointing to the right ventricle.

3) By further caudal tilting the papillary muscles can by imaged as shown below.




right side of the patient
left side of the patient
LV
RV

PSAX papillary muscle level , the papillary muscle level is showing the anterior-lateral papillary muscle(towards the lateral wall) and posteromedial papillary muscle (towards the inferior wall)



Typical PSAX mitral valve level and papillary muscle level :

1) The LV should be Circular in shape and in the middle of the screen in both views, with clear mitral valve(in the case of mitral valve level) centralized within the LV .
2) Permits perpendicular curser over the septum.

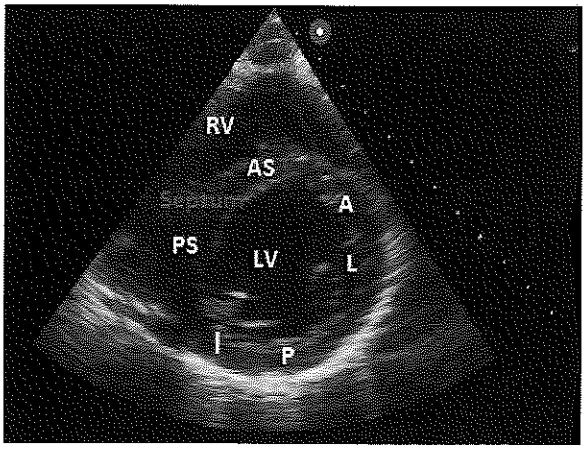
EMSE stepwise approach to optimize the PSAX view at the MV and the papillary muscle levels:

1) Use the pendular motion "rocking motion" to centralize the view in the middle of the screen.
2) the LV should be circular in shape, oblique cut sections will cause an ovoid shape and an overestimation of many calculations as will be discussed later, to correct this ovoid shape try rotational motions in both clock wise and anti clock wise directions until the LV is circular in shape.
3) If the view is still not optimized try to slide (very slowly) in various directions WITHOUT any additional motions (without any rotations, tilting or pendular motions).

According to the desired cut section the probe should be tilted, cranial tilting will bring the great vessels level, further caudal tilting will bring the mitral valve. Further caudal tilting will bring the papillary muscle level.

EMSE stepwise approach to optimize the great vessels level:

1) **Use the pendular motion to centralize the view in the middle of the screen, so that the aorta will be in the middle of the screen.**
2) **To clarify the pulmonary artery fine upward sliding should be applied.**
3) **For further clarification of the pulmonary artery bifurcation further tilting motion could be applied.**
4) **Sliding one space above (keeping the probe orientation as before i.e. the dot towards the left shoulder) will bring a modified view called ductal level view that will be discussed later.**



PS: posterior Septum

AS : anterior Septum

A : anterior wall

L : lateral wall

P: posterior wall

I: inferior wall.

Mnemonic: SALPI

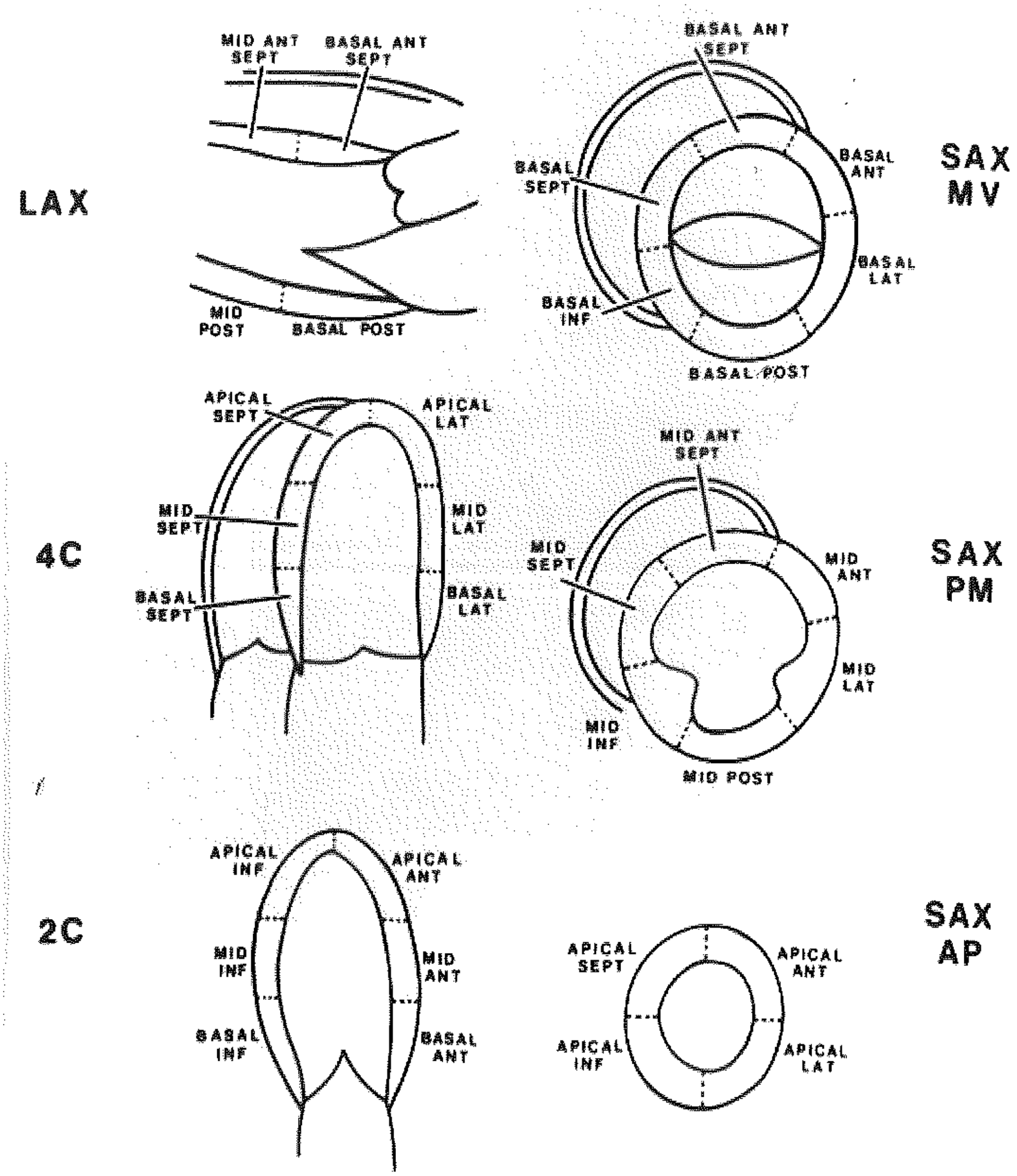
If the mitral valve is imaged then the walls will be basal SALPI (total of 6 basal segments, remember that the septum is divided into two walls: basal posterior septum and basal anterior septum)

If the papillary muscles are imaged then the walls will be mid SALPI (total of 6 mid segments, remember that the septum is divided into two walls: mid posterior septum and mid anterior septum)

If the apical cavity is imaged then the walls will be apical SALI (no posterior wall at the apex) (total of 4 apical segments)

6+6+4 = 16 segments of the heart.

These are the same segments imaged before in the PLAX and the apical views.



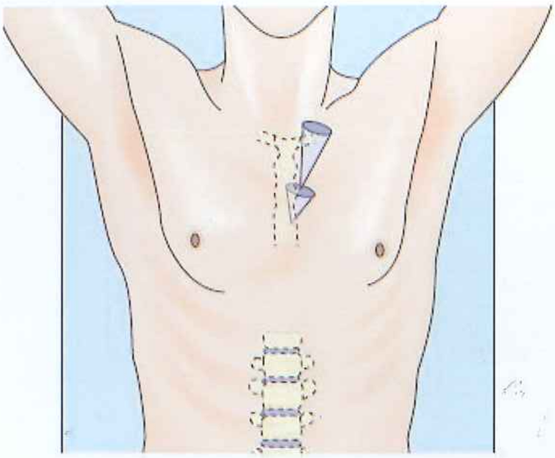
The 16th segment model of the heart.

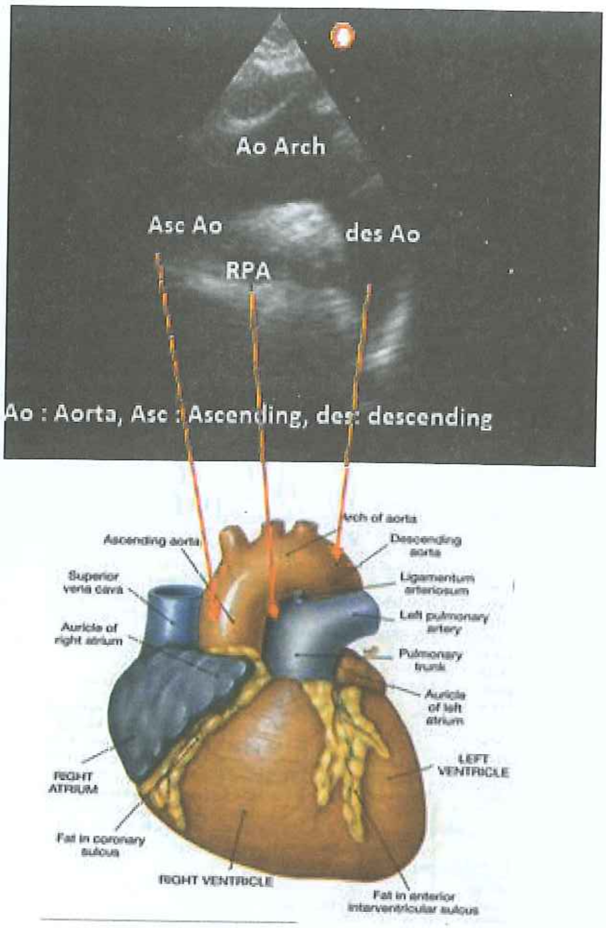
In 2 D imaging, the **EMSE** recommends the use of the 16th model instead of the 17th segment model because all the echocardiography machines are still calibrated on the 16th segment model.

The **EMSE** only use the 17th segment model in speckle tracking and 3 D imaging.

Suprasternal long axis view (SSLA view):

by placing the probe in the suprasternal notch with the dote pointing upwards, apply pendular motion then rotate the probe to direct the dot towards the left jaw, if the view is not optimized try tilting motions to find the view.

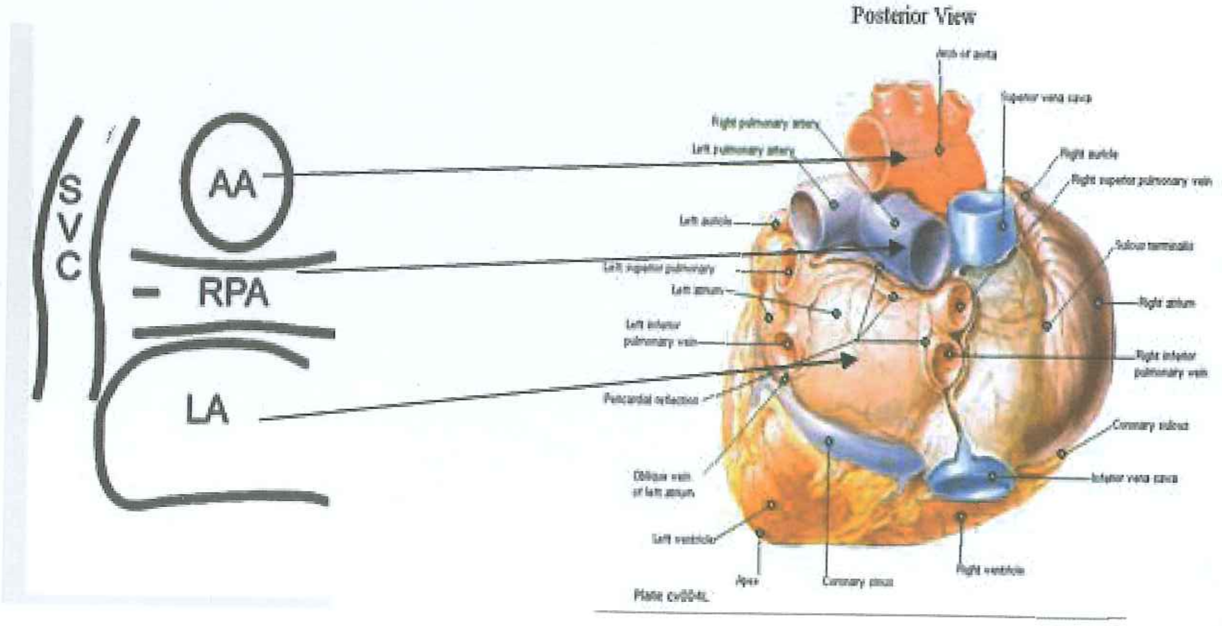


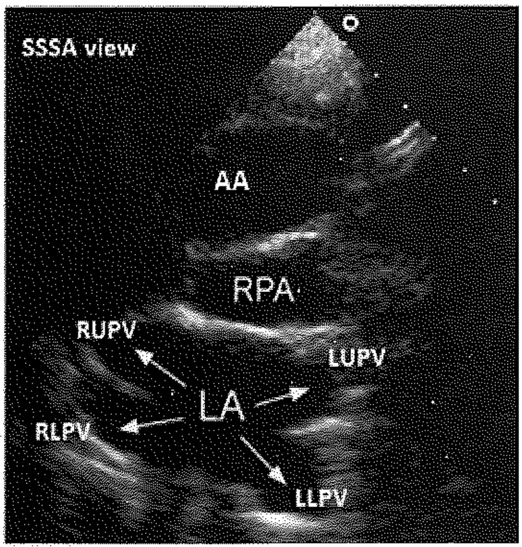


Notice that the right pulmonary artery is below the aortic arch from a supra sternal orientation

Supra sterna short axis view (SSSA view)

From the SSLA view rotate the dot towards the left side. Try to tilt the probe until the view is optimized.

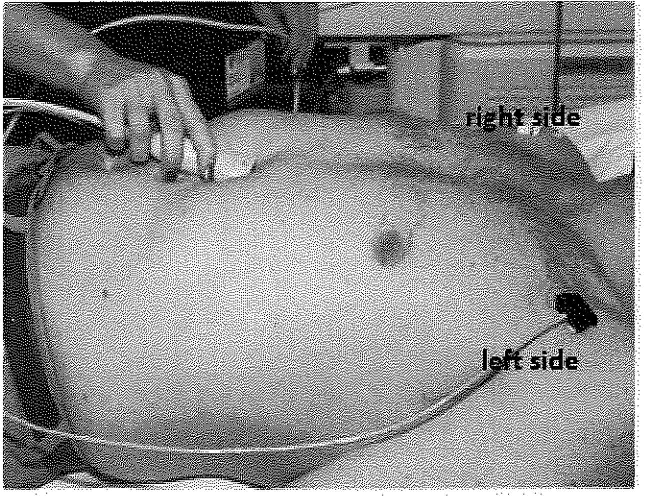




RUPV: right upper pulmonary vein
RLPV : right lower pulmonary vein
LUPV: left upper pulmonary vein
LLPV: left lowetr pulmonary vein

The 4 arrows denote the openings of the 4 pulmonary veins into the LA (correlate the echo view to the anatomical model).

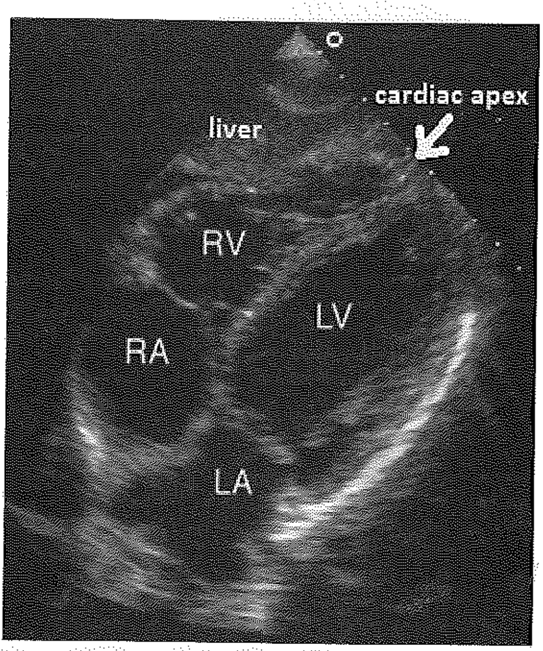
Subcostal views :



Acquiring the subcostal 4 chamber view:

To image the subcostal 4 chamber view, the probe should be placed in the subcostal region as shown. Notice the tilting motion used to direct the beam towards the chest and the heart. The dot is typically directed towards the left side of the patient. By directing the U/S beam in this way, the U/S beam will cut through the 4 chambers of the heart in a longitudinal axis. But remember that the U/S beam will first encounter the liver, so the liver will be the first structure to be displayed up in the screen. By directing the dot of the probe to the left side, the dot is actually directed towards the cardiac apex, so the apex will be displayed towards the dot in the screen.

If the view is not imaged, try upwards/ downwards sliding motions without any other additional motions.



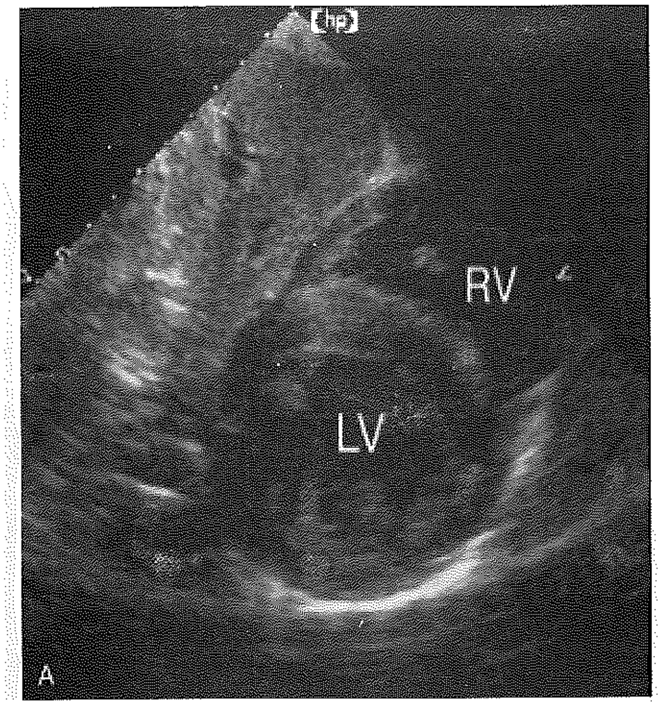
Subcostal 4 chambers long axis view, notice the similarity between this view and the Ap 4ch view. The same cut section, but from a different orientation.

Subcostal short axis orientation:

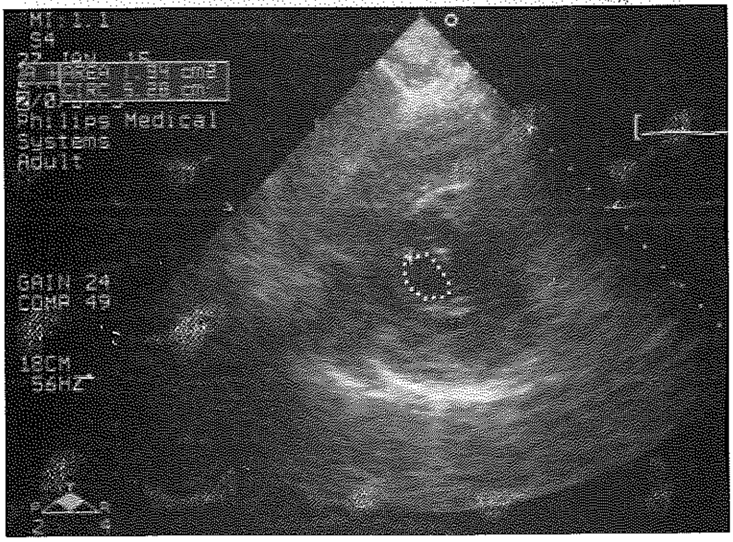
Acquiring the subcostal short axis orientation:

From the subcostal 4 chambers view, rotate the probe in a clockwise manner; **while keeping the U/S beam directed towards the heart;** until the dot is directed towards the left shoulder. Notice that this is the same orientation as the PSAX view but from the subcostal position.

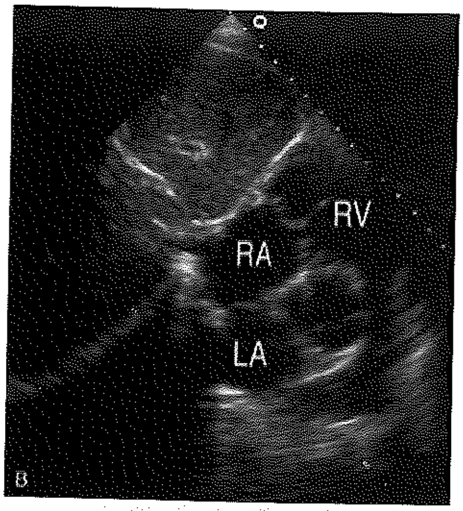
By applying tilting (*this is the same concept of the tilting motion in the PSAX view*) the subcostal short axis view can be sliced to basal, mitral valve and papillary muscle levels (exactly as in PSAX view but from the subcostal position).



Subcostal short axis view papillary muscle level: notice the similarity between this view and the PSAX view.



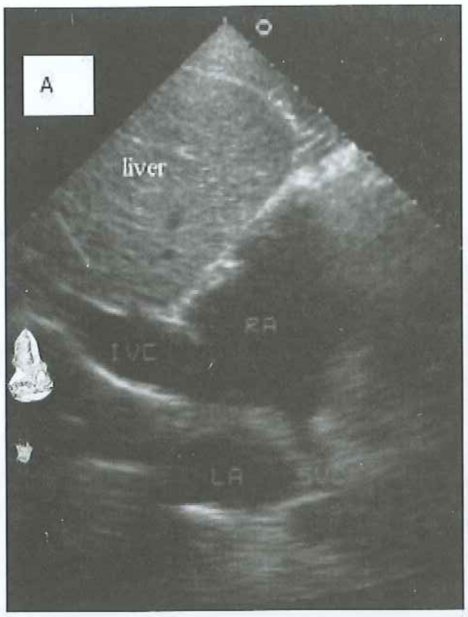
Subcostal short axis view mitral valve level: notice the similarity between this view and the PSAX view. Both views are the same, but from a different orientation (Even MV planimetry can be performed as shown, but this is not the classic view to trace the MV area).

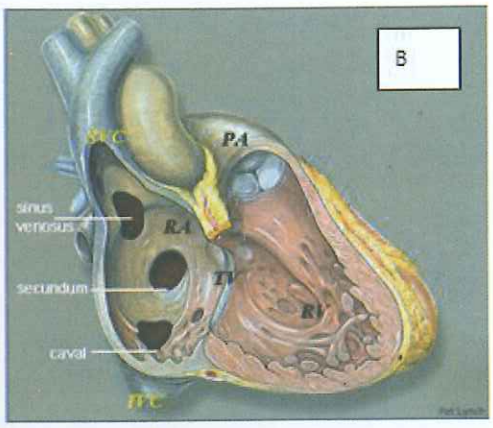


Subcostal short axis view great vessels level (basal level): notice the similarity between this view and the PSAX view.

EMSE practical tip : the main pitfall while acquiring the subcostal short axis view is the false added on pendular motion that will falsely direct the U/S beam towards the abdomen. Also any sliding motions will affect the view.

Subcostal bicaval orientation:





Echo view A: subcostal bicaval view, correlate the echo view with the anatomical cut section.

Anatomical cut section B : this is an anatomical section in the right side of the heart, the model is slightly rotated in an anticlockwise (i.e. slight leftward rotation) to clear out the right side of the heart. The TV (tricuspid valve), part of RA and part of RV are removed to reveal the anatomy of the IAS and the IVS from the right side.

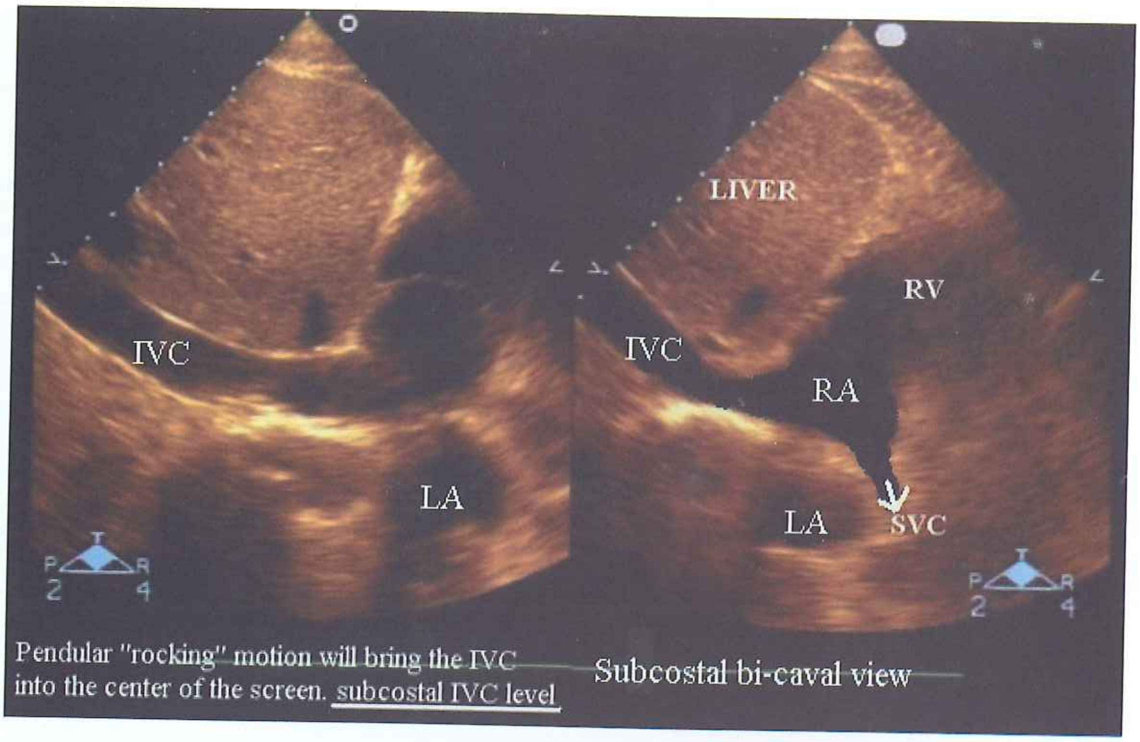
N.B the left side of the heart lies behind this picture.

Remember that the SVC and IVC are not imaged in the Ap 4 ch view.

Notice that there are parts of the IAS related to both SVC and IVC.

As a conclusion there are parts of the IAS (those related to the IVC and the SVC) will not be imaged in the apical 4 ch view. What if there is an ASD in those parts of the IAS ? , those ASDs may be missed. Typically those parts of the IAS which are related to the IVC and the SVC, will be imaged in the bicaval view as shown in the anatomical model.

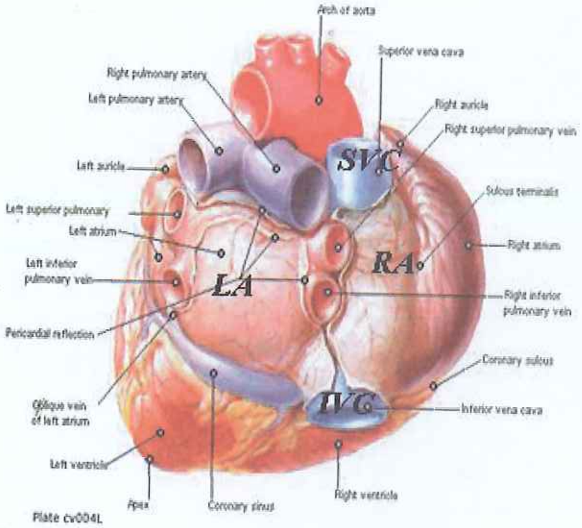
3 defects are shown in the IAS; secundum ASD in the middle of the IAS, sinus venosus ASD SVC type (related to the SVC) and sinus venosus ASD IVC type (related to the IVC). The sinus venosus ASD IVC type is very rare.

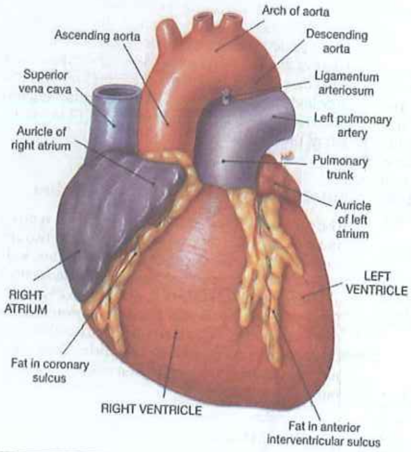


Pendular "rocking" motion will bring the IVC into the center of the screen. subcostal IVC level

Subcostal bi-caval view

Posterior View





Notice that part of the LA actually lies behind the RA.

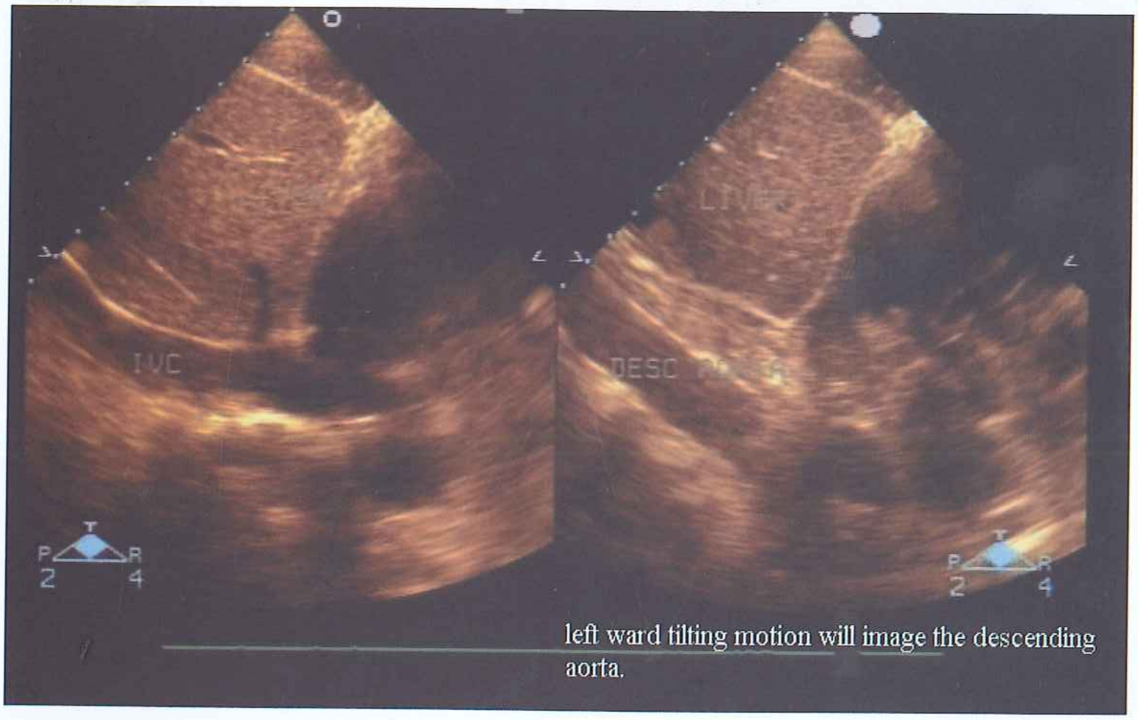
Acquiring the subcostal bi-caval view:

From the subcostal orientation the first structure encountered by the U/S beam is the liver, so the liver will be displayed up in the screen. From the subcostal 4 chamber view position, 90 degrees rotation of the dot of the probe in an anticlockwise manner will bring the U/S beam in a sagittal section with the body.

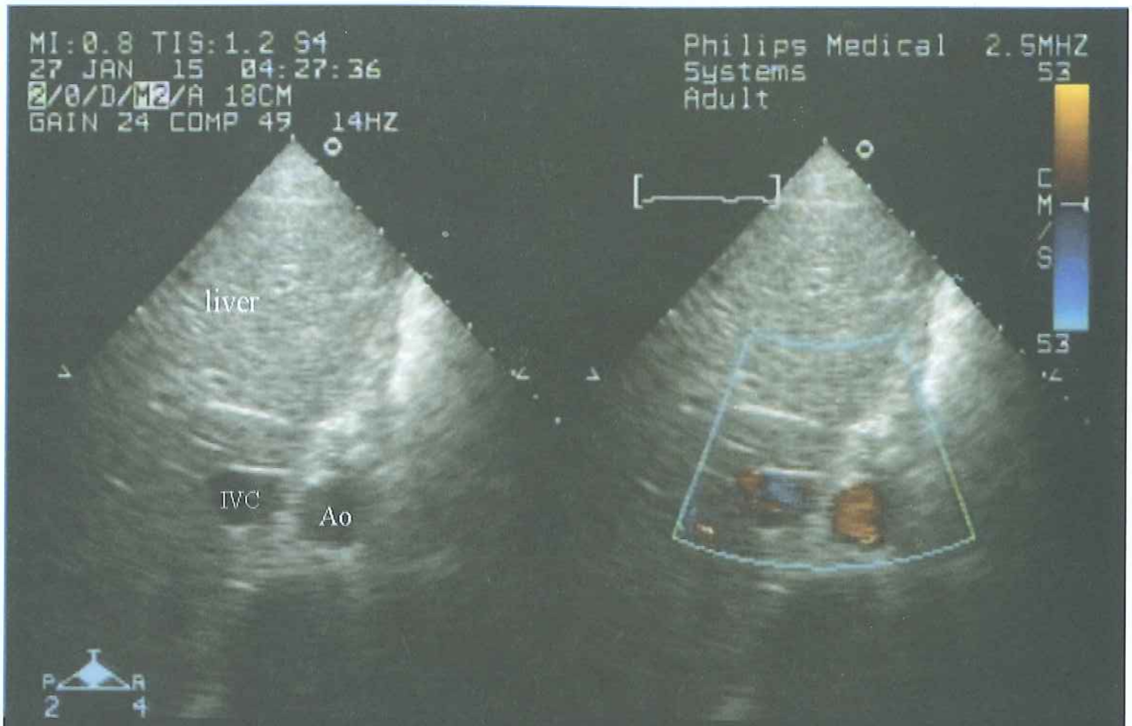
N.B: After rotating the probe, ensure that the beam is still directed towards the chest and not the abdomen.

Using minimal rightwards tilting motions (i.e. the U/S beam will be directed towards the right side of the patient) and minimal rotational motions, the U/S beam will cut through the IVC within the liver, the RA and part of the LA behind the RA. The end result is that the dot of the probe will be directed towards the right shoulder. This will image the part of the IAS related to the SVC and the IVC.

N.B: From this position a leftward tilting motion should image the descending aorta (take care that after this tilting motion the IVC will not be imaged)



left ward tilting motion will image the descending aorta.



Subcostal IVC Aorta short axis view. The U/S beam cuts a transverse section in the subcostal position.

Chapter 2

Clinically applied physics:

Sound waves:

In order to have better imagination and understanding of the ultra sound (U/S) beam, we must understand: what is the sound wave formed of ?

One of the best examples to imagine what a sound wave looks like is to imagine the effect of a vibrating tuning fork in the water, what will happen? A wave front will be propagated! These waves were formed due to the impaction of the tuning fork vibrations into the medium (water) that actually imposed pressure into that medium. As a result of this pressure, areas of peaks & troughs were formed, where the areas of peaks have increased pressure within their particles and the areas of troughs have low "negative pressure "within their particles. Actually the same thing happens when tuning fork vibrates in the air. What will happen in the air? A wave will be propagated exactly as illustrated before with all the changes occurring in the water will occur within the air particles (that are invisible by our ordinary vision). These vibrations (areas of compression and rarefactions of the air particles hit our ear drum and so we can hear the sound).

So now we understand the sound wave. Let's now correlate these waves with the famous mathematical drawing of the sound wave (see diagram 1).

Diagram 1

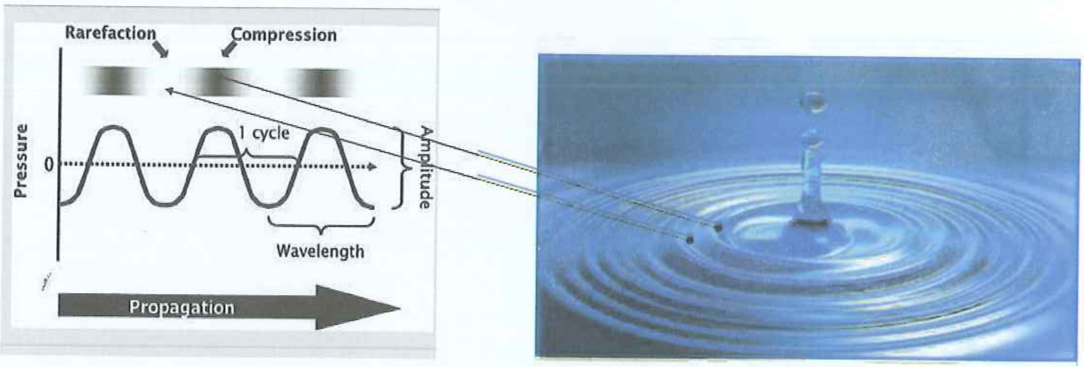


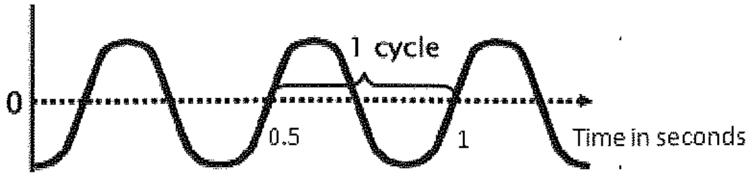
Diagram1

The understandable language for any calculating system is the numbers. So, let's convert these waves to numbers illustrated on a graph. We will simply draw 2 axes, "X" and "Y", then lets illustrate the changes in pressure within the particles on "Y" axis and the change in time on the "X" axis, we will have increasing pressures going up "+ve" on the "Y" axis and going down"-ve" on the same "Y" axis. By drawing this oscillating pressure on the "Y" axis and expressing them against time on the "X"axis, we will have our conventional graph of the wave. (See diagram no. 1)

Wave duration: it is the time needed for a wave to repeat itself.

Frequency: the number of cycles per units of time.

Diagram 1 a Frequency:



In this example the frequency is 2 cycles/ second (simply count the number of cycles in one second) or divide 1 cycle by the period of time of this cycle. It will give the same result.

Frequency = 1/ period F= 1/ 0.5 = 2 cycles per second

So period = 1/ frequency

Cycles per second = hertz

So the frequency is 2 hertz

Wave length

Wave length : it is the distance needed for the wave to repeat itself.

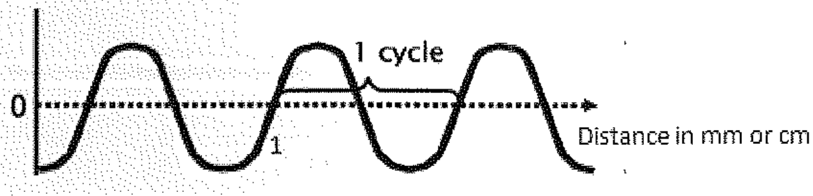


Diagram I b

Speed = distance / time (for example a car is said to move at a speed of 100 km/hour)

Distance is the wave length λ

N.B : Time = period

Speed of sound is constant (C) according to the medium.

So, C= λ/ Period (remember that F = 1/P and the P = 1/ F)

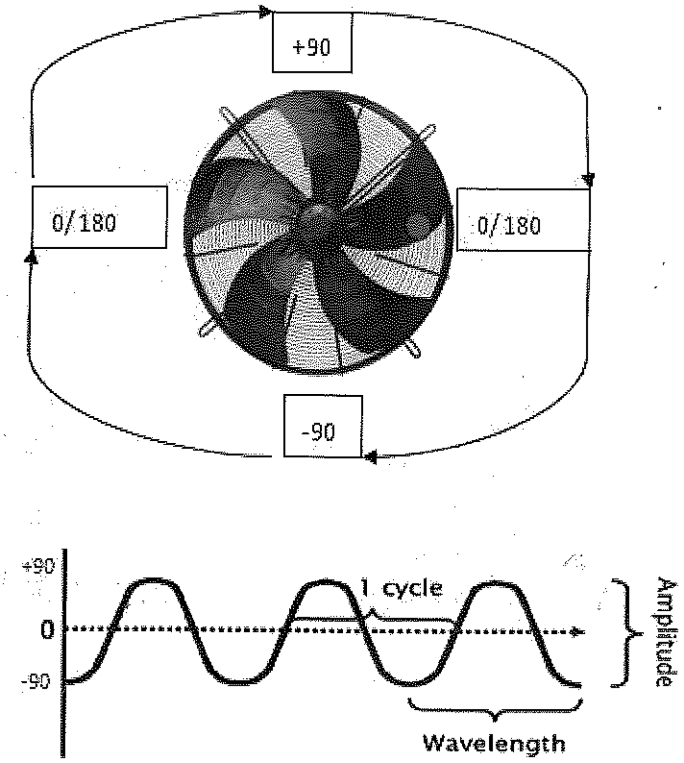
So, C = λ/ (1/F) **therefore: C= λ X F**

So what actually travels in the air are areas of compressions and rarefactions, these areas of compressions and rarefactions are expressed as in diagram no. 1.

Let's see another type of motion that is encountered in our daily life. The idea of understanding a physically seen wave and its transformation to a mathematically illustrated wave is very important to understand sound waves, because nearly the same physics apply to most types of waves, and remember that sound is originally a mechanical wave.

Let's illustrate the motion of a revolving fan in a mathematical manner on an X and Y axis in the diagram below.

Diagram 2



Follow the red dot during the fan rotation, the red dot will start at an angle of 0/180 degrees, after quarter a cycle the red dot will be at an angle -90 degrees, then As the fan will rotate the red dot will be at angle of 0/180 degree, then at +90 degrees then back to the original point at 0/180. By illustrating the degrees on the Y axis we will have one dot oscillating between -90 degree and + 90 degree, by stretching the oscillating motion against time " i.e. the X axis", we will get the same wave as the sound wave, both motion waves are formed of frequencies and wave lengths. The same physics that apply to the sound waves will apply to the waves of the rotating fan.

If the fan completes one full rotation in one second, the frequency is said to be "one cycle/ second". If the fan completes 2 full rotations in one second, the frequency will be 2 cycles/ second, and so on. If the fan rotates slower, for example : only quarter a cycle in one second, the frequency will be ¼ cycle/ second, and if the fan rotates half a cycle in one second, the frequency will be ½ cycle/ second.

The General Concept of Aliasing:

Why do car wheels look like they are spinning backwards at high speed?

The motion of a slowly rotating wheel or a fan is seen in the true direction; for example a slowly clockwise revolving fan is seen turning in a clockwise manner; but what will happen if the speed of this fan is increased? At a certain speed you will see the motion of this fan as if rotating in the opposite direction (See diagram 3). Did the fan actually revolve in the opposite direction? No, it did not, but your eyes falsely perceived the motion in the opposite direction. **This is called Aliasing**.

Diagram no. 3 showing aliased rotation. At high speeds, we may see the motion in the opposite direction; this is due to the very fast frequency of rotation that is more than our optic nerve firing.

See text for details.

Step by step understanding of aliasing:

Imagine that our optic nerve fires only one nerve impulse to the brain per second. Then the pulse rate frequency (***PRF)*** of the optic nerve is said to be 1 pulse per second.

In the following examples: ***(critically important information.)***

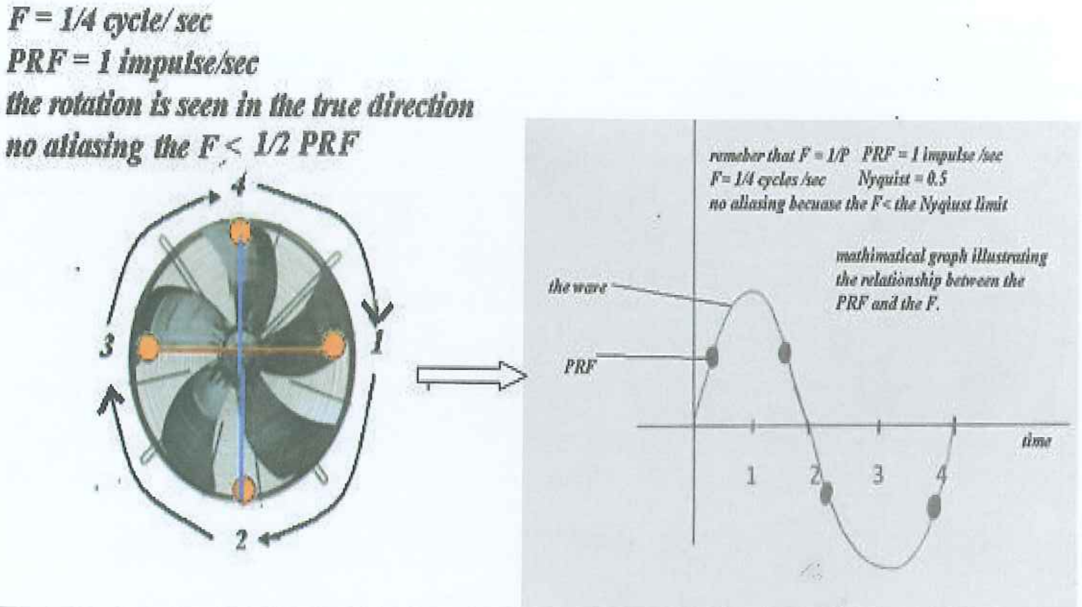
- The PRF = 1 impulse /sec
- The half value of the PRF is called the Nyquist limit (Nyquist is the name of the scientist who discovered this value). **Nyquist = ½ PRF**
- The frequency is **F.**

N.B the conclusion that we want to reach in the following discussion is that aliasing will occur when the F is > Nyquist limit. i.e. when the F > ½ PRF.

If the fan is rotating in a clockwise direction at a rate of ¼ cycle / second, (remember that we are imagining that the optic nerve sends only one impulse per second to the brain, See diagram no 4).

Starting from position no.1, after one second our brain will see the dot at position 2. After one second we will see the dot at position 3. After another second we will see the dot at position no.4. Then after another second the dot will be back to the original position i.e. position no.1 . So, the brain will collect all the four **frames** together in the right sequence position 1 then position 2 then position 3, then position 4, then position 1 again and we will see the fan rotating in the true "real" clockwise direction.

Diagram no. 4



F = 1/4 cycle/ sec
PRF = 1 impulse/sec
the rotation is seen in the true direction
no aliasing the F < 1/2 PRF

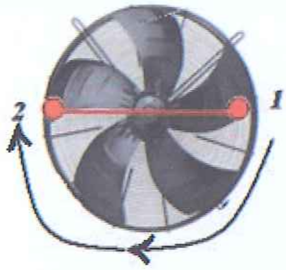
4
1
2
3
remeber that F = 1/P PRF = 1 impulse /sec
F= 1/4 cycles /sec Nyquist = 0.5
no aliasing becuase the F< the Nyqiust limit
mathimatical graph illustrating the relationship between the PRF and the F.
the wave
PRF
time
1
2
3
4

Starting from position no.1, after one second our brain will see the dot at position 2, then after another one second we will see the dot at position 3, After another one second we will see the dot at position no.4, After another second the dot will be back to the original position i.e. position no.1 . So the brain will collect all the four **frames** together in the right sequence position 1 then position 2 then position 3, then position 4, then position 1 again and we will see the fan rotating in the true "real" direction. No aliasing occurred because the F is less than ½ the PRF (remember that the PRF is imagined to 1 impulse /second), **i.e. the F < the Nyquist limit →no aliasing.**

N.B : The red dots are equivalent to the black]k dots

If the fan is rotating in a clock wise direction but in a faster rate, for example, by a rate of ½ cycle / second. Starting from position no. 1, after one second we will see the dot at position no. 2, then after another second we will see the dot at the original position "position no. 1" back again. At this situation our brain will collect the frames together, position 1, then position 2, then position 1 again, and so on. So we will see the fan rotating in the true "real" clockwise direction. At this situation the frequency = ½ cycle per second, and the optic nerve PRF is 1 impulse/second. i.e. the frequency is ½ the PRF (the frequency = Nyquist limit) and our brain is still collecting the frames in the true sequence, which is position no.1 then 2 and we still see the rotation of the fan in the true direction, i.e. no aliasing. See diagram no 5.

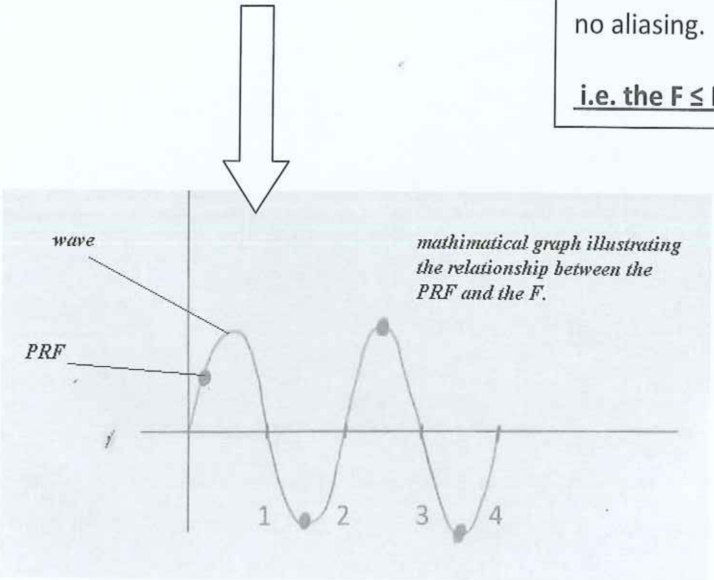
Diagram no 5



F = 1/2 cycle/ sec
PRF = 1 impulse/sec
the rotation is seen in the true direction
no aliasing, the F is less than or equal to the half of the PRF value

From position no. 1, after one second we will see the dot at position no. 2. After another second we will see the dot back again at the original position i.e. "position no. 1". At this situation our brain will collect the frames together, in the following sequence → position 1, then position 2, then position 1 again, and so on. So we will see the fan rotating in the true "real" clockwise direction. At this situation the frequency = ½ cycle per second, and the optic nerve PRF is 1 impulse/second. i.e. the frequency is ½ the PRF and we still see the rotation of the fan in the true direction, i.e. no aliasing.

i.e. the F ≤ Nyquist limit → no aliasing



mathimatical graph illustrating the relationship between the PRF and the F.

F = ½ cycle/ sec

PRF = 1 impulse / sec

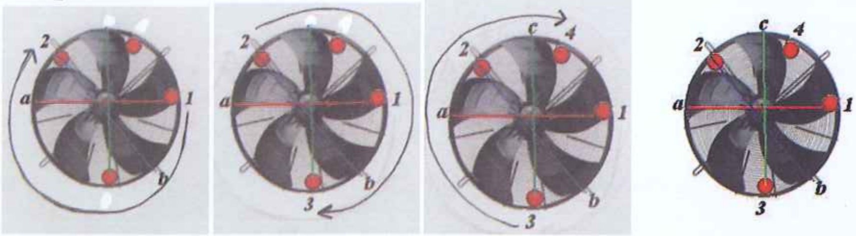
Nyquist = 0.5

So the F= Nyqiust

Therefore no aliasing will occur

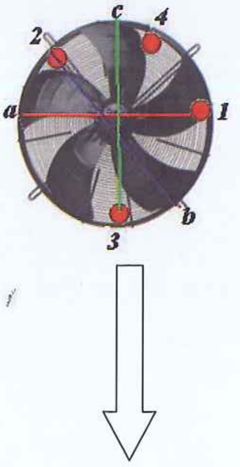
Let's see what will happen if the fan rotates by more than ½ cycle/ second (see diagram no 6), and still the optic nerve PRF is one impulse / second, i.e. the frequency is more than ½ the PRF. Let's start from position no 1, so, after 1 second we will see the dot at position 2,*(if the frequency was ½ cycle per second the dot would have been seen in position "a", but as the F is more than half cycle /sec so the dot will be seen a little bit farer than position "a" i.e. at position 2).* Then after another one second the dot will be seen at position 3,*(if the frequency was ½ cycle per second the dot would have been seen in position "b", but as the F is more than half cycle /sec, the dot will be seen a little bit* *farer than position "b". i.e. at position 3).* , then after another one second we will see the dot at position 4,*(if the frequency was ½ cycle per second the dot would have been seen in position "c", but as the F is more than half cycle /sec, the dot will be seen a little bit farer than position "c". i.e. at position 4).* So the brain will collect those frames in the following order position no.1, then position no. 3, then position no 2 , then position no 4. The result will be that we will see the fan rotating in the true direction (frames 1 & 2) but also some times we will see it rotating in the opposite direction (frames 2 & 3), i.e. aliasing occurred and we will perceive the velocity " falsely" in an opposite direction.

Diagram no 6



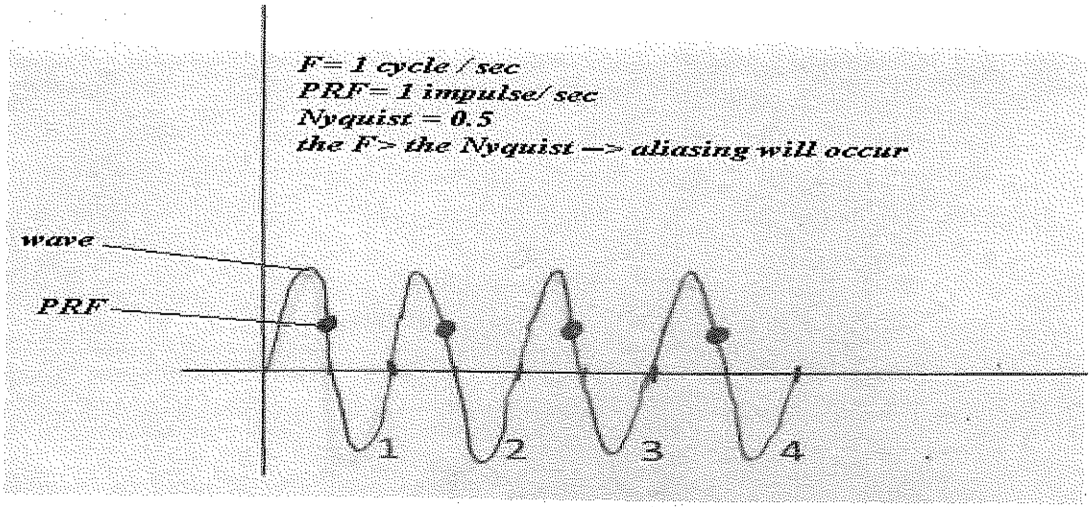
The brain will finally collect the frames in the following order 1, 3, 2, 4 → false frame analysis that will distort the rotation direction.

F= 3/4 cycle /sec
PRF= 1 impulse/sec
the rotation will be seen in the false direction and some times in the true direction. i.e. aliasing occured when the F > 1/2 PRF



Let's start from position no 1, then after 1 second we will see the dot at position 2, then after another second the dot will be at position 3, then after another second we will see the dot at position 4. So the brain will collect those frames in the following order position no.1, then position no. 3, then position no 2 , then position no 4. The result will be that we will see the fan rotating in the true direction but also some times we will see it rotating in the opposite direction, i.e. aliasing occurred and we perceived the velocity " falsely" in an opposite direction.

So aliasing occurred when the F > ½ PRF.



As a conclusion, we perceive the rotation in the wrong direction only when the F > Nyquist limit. (I.e. F more than the half value of the PRF)

Actually our optic nerve fires about 60 impulses per second to the brain, so the real PRF (pulse rate frequency) of our optic nerve is 60 impulse per sec. so the Nyquist limit will be 30. Therefore, any perceived frequency (rotating fan) that will be less or equal to the Nyquist limit will be seen in the true "real "direction. I.e. any frequency less than 30 cycles per sec will be perceived in the true direction and no aliasing will occur. On the other hand, any frequency that will be more than the Nyquist limit will be seen in the false direction. I.e. any frequency more than 30 cycle per sec will be perceived in the false direction and aliasing will occur.

So, as a general rule in physics: aliasing will occur when any pulsatile receptor receives a frequency that is more than the Nyquist limit of the receptor.

Let us illustrate the previous data in a mathematical way, diagram no 7.

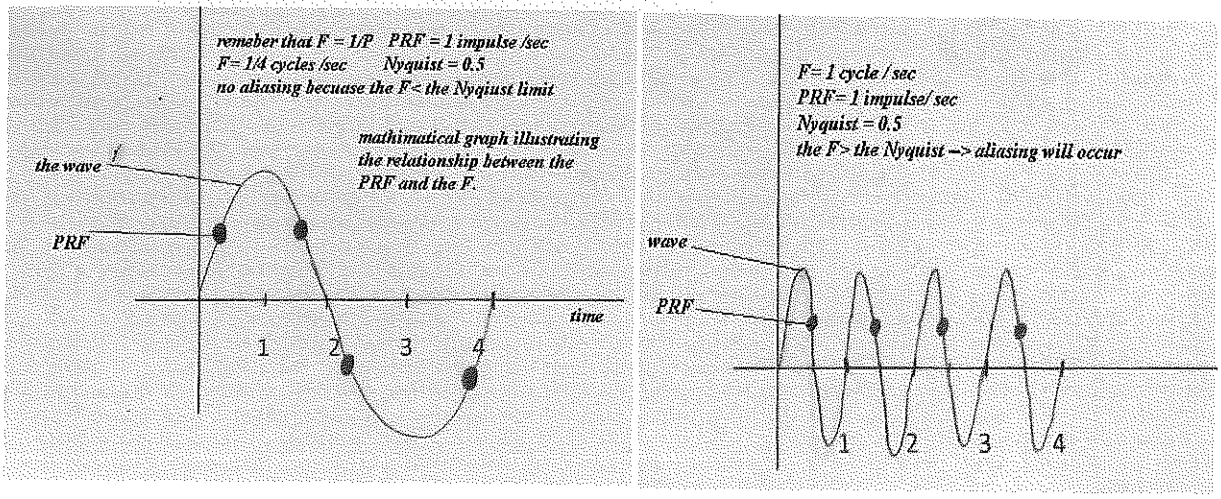

remeber that F = 1/P PRF = 1 impulse /sec
F= 1/4 cycles /sec Nyquist = 0.5
no aliasing becuase the F< the Nyqiust limit
mathimatical graph illustrating the relationship between the PRF and the F.
the wave
f
PRF
time
1
2
3
4
F= 1 cycle / sec
PRF= 1 impulse/ sec
Nyquist = 0.5
the F> the Nyquist —> aliasing will occur
wave
PRF
1
2
3
4

Diagram no 7

The waves illustrated represent the rotating fan, Remember from diagram no 2 the mathematical representation of an oscillating fan.

The black dots represents the PRF of the optic nerve.

Calculate the frequency (F= 1/P, or count the number of waves in 1 sec), count the PRF and calculate the Nyquist limit (Nyqiust is ½ PRF), at time the F> Nyquist →aliasing occur.

We will apply the same concept later on while discussing aliasing in U/S.

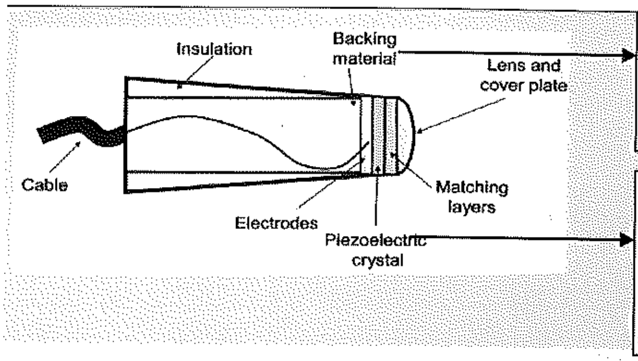
The rate of firing of the nerve impulses is equivalent to the pulses fired from the probe. (See later.)

The wave of the fan is mathematically equivalent to the sound wave. (See before)

Chapter 3: How does the machine work?

In order to understand the image orientation, pitfalls and measurements we must understand some physics, otherwise it will be very difficult to accept and memorize a lot of important aspects.

The probe contains what's called piezoelectric crystals. These are very unique substances which produce sound waves (U/S beam) when they are subjected to an electric current , then these unique piezoelectric crystals can generate voltage when the sound waves (U/S beam) reflects back to it. (SEE DIAGRAM no 8)



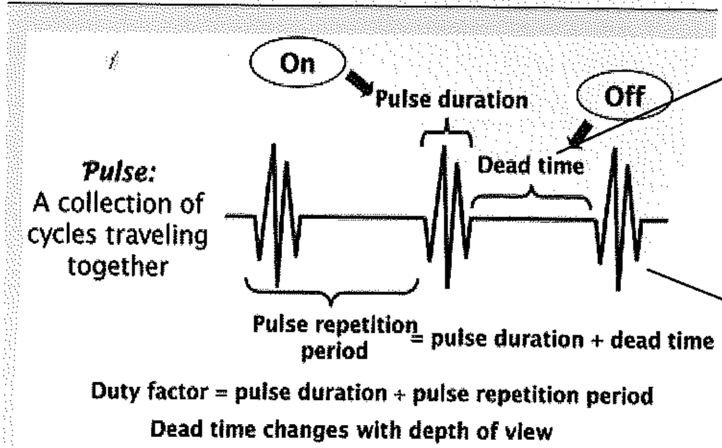
Prevent the ringing response. i.e. prevents that the U/S waves reflects back to the body.

Either produces the U/S beam when subjected to electric current, or generates electric current when it receives a reflected U/S beam

Diagram no 8

In general, the probe can send the sound waves (U/S beam) in two main forms either in a single line interrogation or in a many lines " fan like projection" , then when the sent waves reflect back from the various body structures, the probe can definitely sense them and turn them back to voltage(the understandable language by the computer). Also the probe can send the U/S beam in a pulsatile manner (2D imaging and Pulsed wave and color Doppler) or a continuous form (continuous wave Doppler). See diagram 9.

Now we have voltages in our computer system that correspond to waves. These different voltages can be handled by the computer by TWO different equations, the range equation and the Doppler shift equation.



On
Pulse duration
Off
Dead time
Pulse:
A collection of cycles traveling together
Pulse repetition period = pulse duration + dead time
Duty factor = pulse duration ÷ pulse repetition period
Dead time changes with depth of view

Dead time "listening time" in which the piezoelectric crystal can receive back the U/S beam reflected from various structures

These are pulses of U/S waves that are generated from the piezoelectric crystals within the probe

Diagram 9.

Every piezoelectric crystal can either receive U/S beam or send U/S beam, but the single piezoelectric crystal cannot perform a dual action of sending and receiving U/S beam ***at the same time !!.*** i.e. the piezoelectric crystals either receive voltage from the machine (via the wire of the probe) that cause them to vibrate and produce U/S beam into the body, or receive U/S from various reflectors that cause conformational changes in the piezoelectric crystals that generate voltage that will be carried (via the wire of the probe) back to the machine.

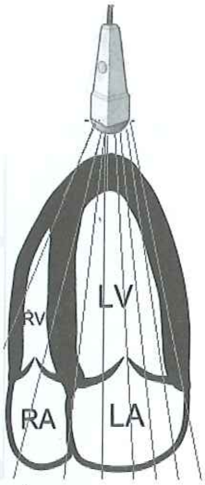
THE RANG EQUATION: this is the equation that controls the concept of the 2D imaging and depends mainly on the fact that the probe will send U/S beam in a pulsatile manner. The simple concept of the range equation is that the first structure that the beam will hit will be displayed first on the top of the screen " near zone " and the last structure the beam will hit will be lastly displayed down the screen "far zone" , ex: when placing the probe in an apical position the first structure ;by logic ; just beneath the probe is the apex so the apex is displayed first on the top of the screen "near zone", and the last structure displayed down the screen "in the far zone" will be the base of the heart , thus forming the classic apical 4 chamber view.

See diagram 10. So, by knowing the speed of U/S in soft tissue and blood, and by being able to calculate the time interval that elapses between the ***pulsatile*** beam's departure from the probe to its ***pulsatile*** return back to the probe , we can simply calculate the distance to that reflector : speed = distance / time . So the distance = speed x time. This calculated distance is the distance of the whole journey the beam took ;what's called the Go- Return flight; so if the computer wants to calculate the distance to the reflector it will have to divide this number by 2. (consider that every pulse represents a car, if we know the speed of this car and the time that this car spent in the Go- return journey, then we can easily calculate the distance traveled by this car)

The speed of sound is fixed according to the medium (1540 m/s, 1.54mm/microsecond) so, the distance to the reflector = 1.54 x 1/2time of the flight, which is the same as 1.54/2 x time of the flight (0.77x time of the flight).

So in the 2D imaging modality the probe sends the beam waves in a pulsatile multiple lines "fan like projection" and then receives the reflected beam waves in a pulsatile manner too, (instantly calculating the Go- Return time) and introducing these information to the Range equation, thus calculating distances. But it is mandatory to remember that every dot on the screen was originally a timing that is transformed to distance. I.e. every dot is understood in terms of time to the machine and is understood in terms of distances (cm) to doctors.

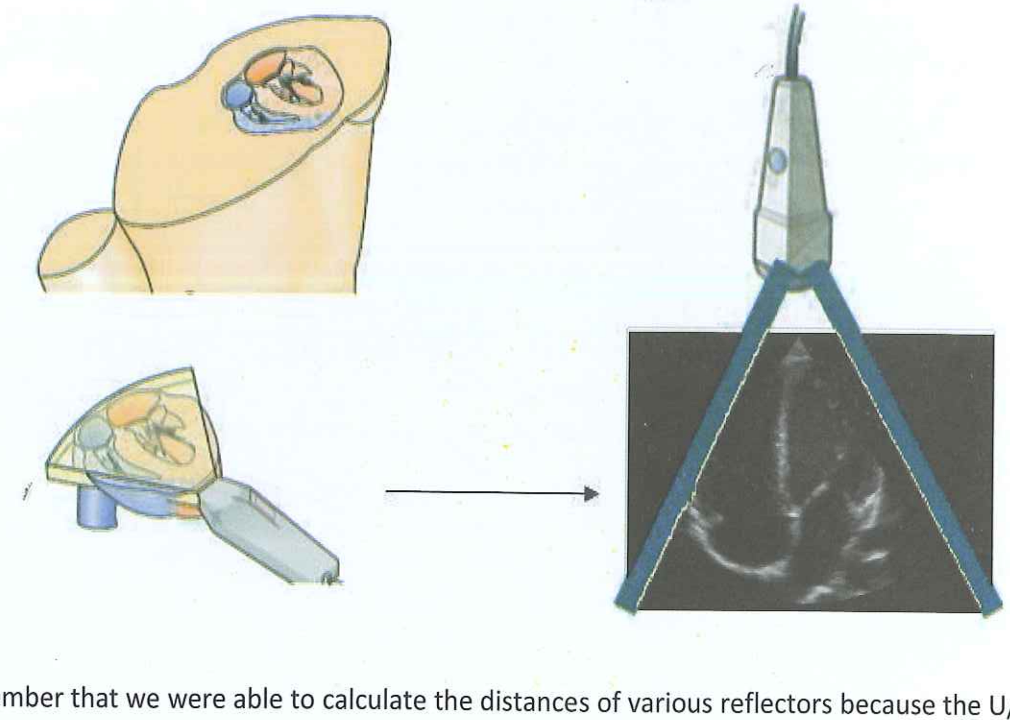
Example :
the probe sends many impulses in many scan lines, part of these impulses energies are reflected back to the probe, and part of the impulses energies penetrates into the body, so these impulses will be reflected back to the probe at different timings, i.e. those impulses that reflects back from the apex will reflect in short time, and those impulses penetrating into the body to be reflected from a more far reflector will be reflected back to the probe in relatively a longer time, i.e. reflectors from the base of the heart while imaging from an apical position.
for example C= D/ T, so D = C X T
1.54 mm/us = Distance of the reflector/ time of the flight of the U/S beam
time of the flight is defined as the time that elapses from the beam's departure to the beam reciving,
so. as the apex is a near reflector the time of the flight is "for example" 10 u.s , so the go-n return distance will be 10 u.s X 1.54 mm/u.s, D= 15.4 mm, i.e.1.54 cm. so the distance to the reflector will be half that value, i.e. 0.77 cm from the probe, which is a relatively very near reflector



each sacn line represents one impulse.

diagram 10

The imaginary place of the probe over the screen



Remember that we were able to calculate the distances of various reflectors because the U/S beam was generated from each single crystal in a pulsatile manner and received by each single crystal in a pulsatile manner too (the crystals receives back the reflected U/S beam in the listening time "see before"). So the computer calculates the exact timing that elapsed between the pulse firing and receiving of each single crystal. This means that the computer can calculate the time of the flight that the U/S beam travelled from firing to receiving, and as discussed before it can simply calculate the distances as the D = C * T (D= the ultrasound go-return distance , C= 1.54mm/us speed of U/S, T= time of the flight)

What will happen if the probe sends the U/S wave continuously instead of being pulsatile? To do this one piezoelectric crystal should fire but another crystal must receive (the crystals cannot perform dual action, see before). This will make the computer unable to calculate the time that elapsed between each firing and each receiving because first, the firing crystal is different from the receiving one, and second the firing is continuous rather than pulsatile. This means that there is no time between firing and receiving of the impulses (there is no impulses from the first place!! It is a continuous wave)

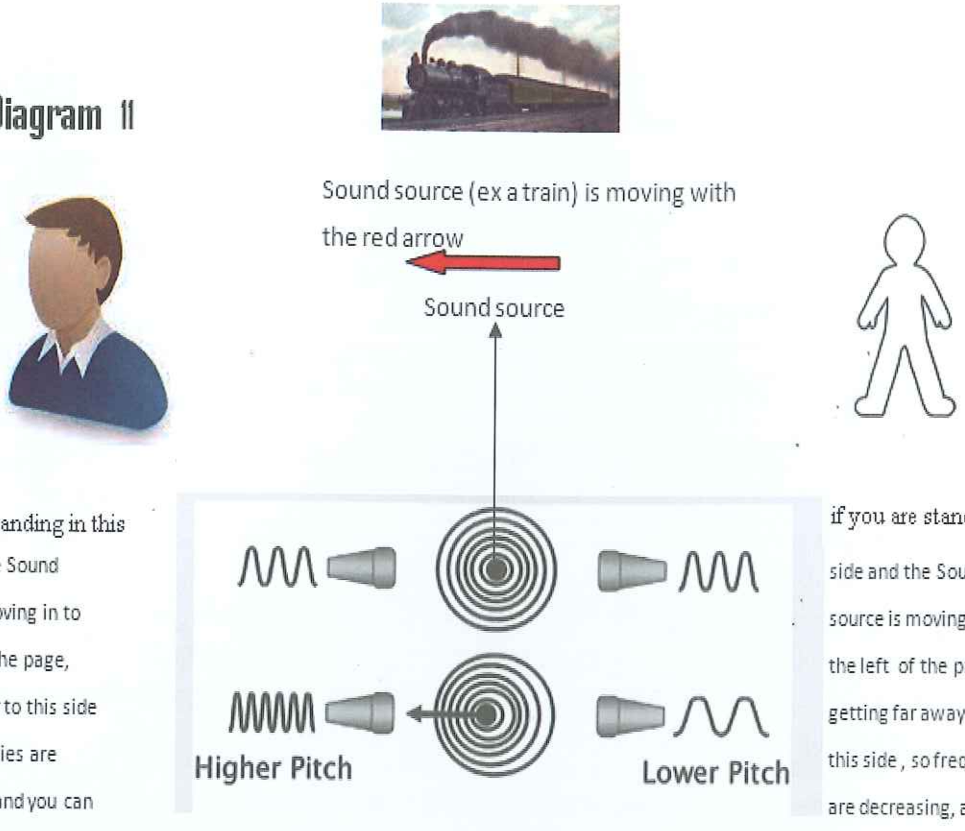
As a conclusion any pulsatile system can localize the distances while any continuous system cannot localize the distances of various reflectors.

DOPPLER SHIFT EQUATION "frequency shift equation":

The popular Austrian physicist; Christian Doppler; had discovered that the when a sound source approximates a certain listener the frequency of this sound wave increases. Conversely, as the sound source moves away from a certain listener, the frequency decreases. He transformed this principle into the frequency shift equation, which is known as the Doppler shift equation. In the Doppler shift equation, the probe sends and receives the U/S waves (either in a continuous or a pulsatile manner; see later), but now it is concerned with frequencies rather than timings of the returning U/S.

To understand the concept of this equation, you must first understand how actually your brain understands sounds as being coming close or getting far away. You must understand that this is a matter of frequencies. See diagram 11 and 12 a, b, c.

Diagram 11



if you are standing in this side and the Sound source is moving in to the left of the page, getting near to this side ,so frequencies are increasing, and you can realize that the train is travelling towards you.

if you are standing in this side and the Sound source is moving in to the left of the page, getting far away from this side , so frequencies are decreasing, and you realize that the train is travelling away from you

The brain actually calculates the shift (differences) in frequencies, and from this shift the one can understand two important information: A) that the sound is coming forward or getting away, and B) the speed of the object transmitting this sound. This is the concept of the frequency shifts known as Doppler shift. See also diagram 12.

But inside the heart there are no an ambulance cars nor trains!.

Rather the probe sends an U/S beam in a single line integration. The sonographer directs this single line (cursor) into the blood flow in a form that this beam will hit the blood flow. Now the beam will reflect from the blood flow as if it had been sent from the blood flow itself, (as if there is an ambulance car or a train coming towards the probe). High velocity flow will cause the beam to reflect with high velocity and thus leading to high frequency shifts, while low (normal) velocity flow will cause the beam to reflect with low velocity leading to low frequency shifts. See diagram 12

If the flow is directed towards the transducer, the frequency shifts (Doppler shifts) will be +ve, and if the flow is directed away from the probe the frequency shifts will be –ve.

+ve Doppler shifts will lead to a calculated +ve velocities , while –ve Doppler shifts will lead to a calculated –ve velocities.

Thus the machine knows the frequency sent (determined by the manufacturer). Then it calculates the returning frequencies by what's called fast Fourier Transformer. And by introducing all this information to the Doppler shift equation (see diagram), it can calculate the velocity.

Doppler shift equation

$$\Delta F = 2\ V\ F_o / C$$

$$V = \Delta F .\ C / 2\ F_0$$

ΔF (delta F) is the difference between the transmitted frequency (from the probe) and the received frequency (by the probe)

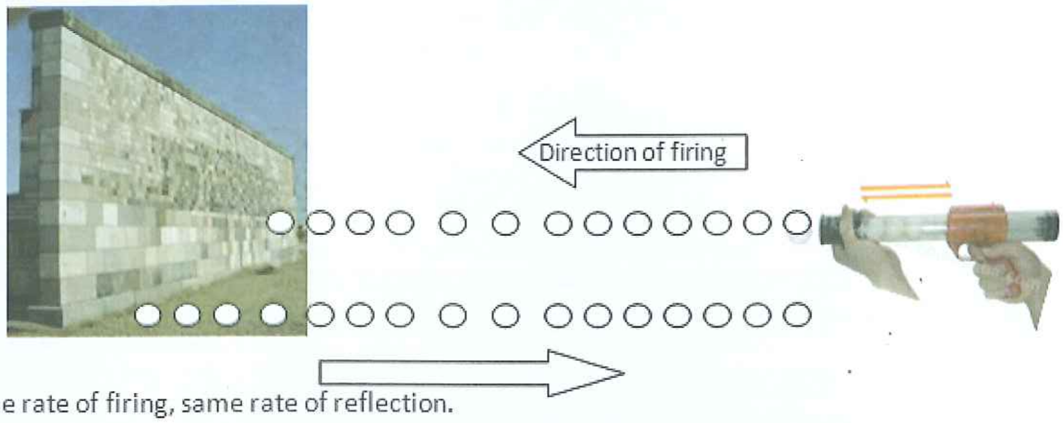
V is the required velocity of the blood (remember that as the U/S beam hits the flow it will be reflected from it as if it was transmitted by the flow)

F_o is the original frequency sent by the probe (determined by the manufacturer)

C is the speed of sound (1540 m/s)

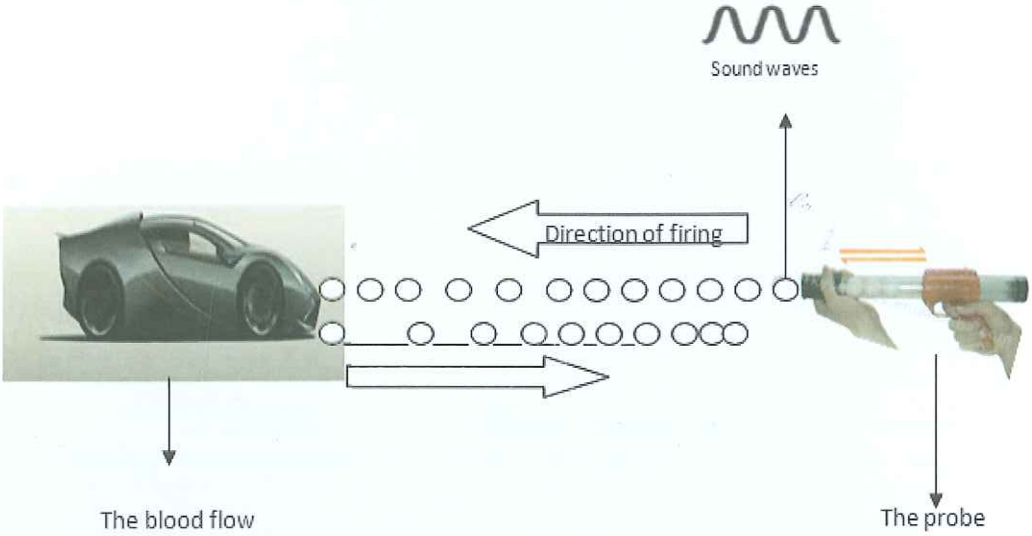
The resultant velocity "V" will be multiplied by cosine theta. Theta is the angle of incidence between the U/S beam and the blood flow. It should be zero, and cosine zero equals 1.

Diagram 12 a : simplified concept of the Doppler/ frequency shift to calculate the velocity

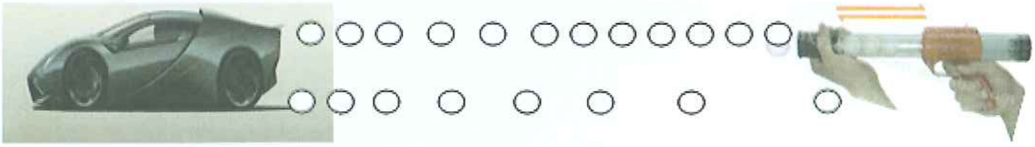


Same rate of firing, same rate of reflection.

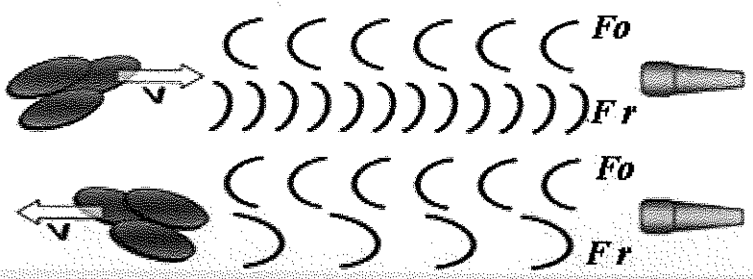
By sending these balls at a known rate with known velocity, the balls will be reflected back with the same rate. (Fixed object) (This equivalent to a zero Doppler shift)



By firing these balls at a known rate with known velocity, If the car is moving towards the Gun at a given velocity (v), the reflected rate (F_r) will be higher than the emitted firing rate (F_0). If the car is moving away from the transducer, the opposite will occur. (See below)



By sending these balls at a known rate with known velocity, the balls will be reflected back BUT with a different rate. If the care is travelling away from the gun the rate of returning back will be decreasing (equivalent to a -ve Doppler shift).



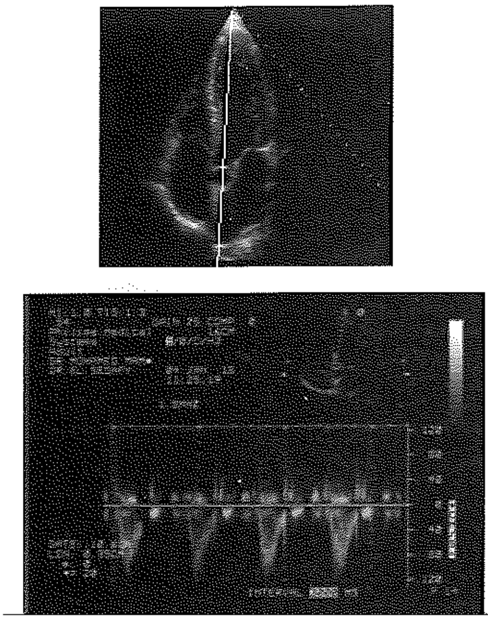
same concept is applied to blood flow. If the red blood cells are moving toward the transducer at a given velocity (v), the reflected frequency (F_r) will be higher than the emitted frequency (F_0). If the red blood cells are moving away from the transducer, the opposite will occur.

diagram 12 b

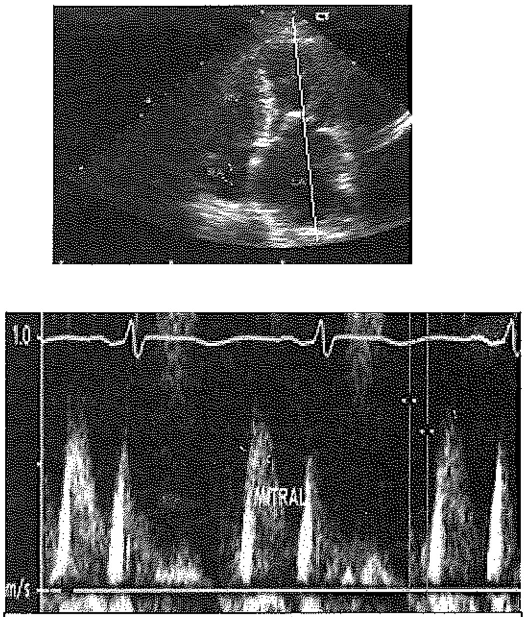
Step by step understanding of the final Doppler signal demonstrated on the echocardiography machine:

1) The echocardiographer aligns the U/S beam parallel to the blood flow.
2) Then by pressing the PW / CW button, the probe sends the U/S waves with a known frequency (F_o), then perceives them in different frequency (F_r), (the probe can calculate the received frequency by Fast Fourier Transformer). All these data are sent to the computer system of the machine, so the computer calculates the delta F "ΔF " ($\Delta F = Fo - Fr$). Then the computer calculates the equation as: $V = \frac{\Delta\,F.\,C}{2\,Fo}$. The resultant velocity will be demonstrated over graph where the X axis is the time and the Y axis is the velocity. In the following fraction of a microsecond, the probe sends again the U/S beam with the same frequency and it will perceive back the reflected U/S wave in another frequency (either in the same, more or less Frequency), then introduces all the information again to the machine to calculate the velocity which will be demonstrated on the same graph. The whole process will be repeated a microsecond after a microsecond. The result is a plotted graph for the change of velocity against time.

N.B : The probe can calculate the received frequency by Fast Foruee Transformer



Probe placed over the apex and the curser line "U/S beam" is Aligned over the aortic valve, the normal blood flow is directed from the LV to the aorta. i.e. away from the probe, so the returning frequency shifts are –ve



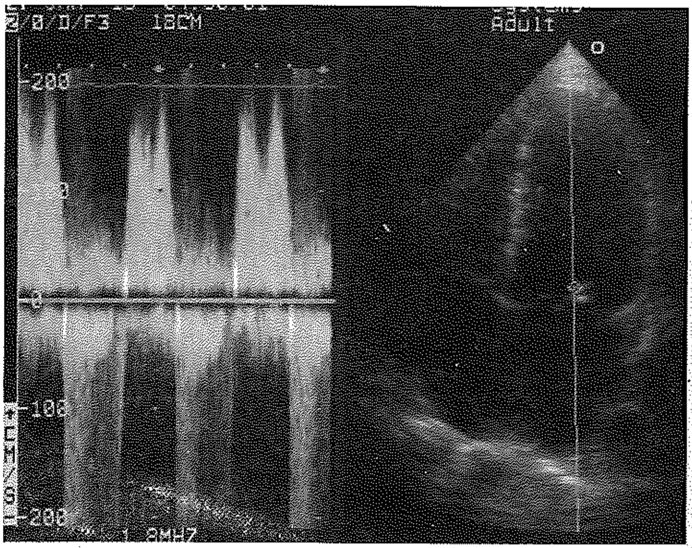
Probe placed over the apex and the curser line is Aligned over the mitral valve, the normal blood flow is directed from the LA to the LV, i.e. towards the probe, so the returning frequency shifts are +ve leading

Diagram 12 C

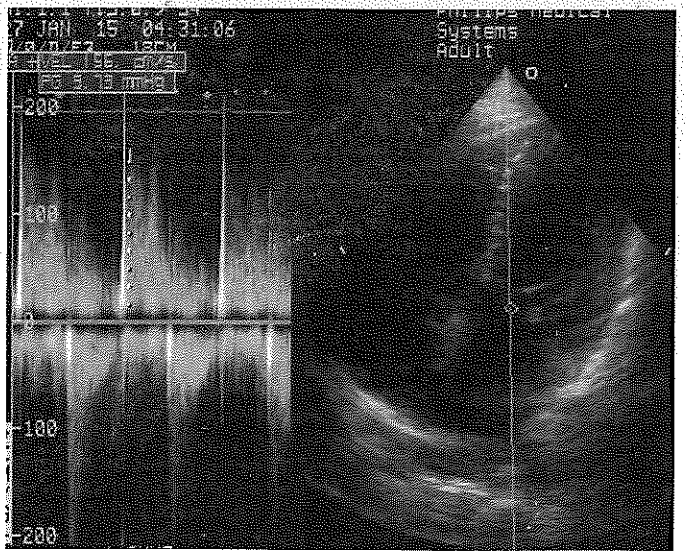
The angle of incidence:

However for the Doppler equation to be accurate, the U/S beam must encounter the blood flow in a parallel fashion, i.e. the angle **"angle theta"** between the blood flow and the U/S beam is zero, so the U/S beam can be hit by the full velocity of the blood flow. Rather if it encounters the blood flow with angulation, the reflected beam velocity will be underestimated proportional to the value of the cosine of the angle made between the U/S and the blood flow. So the resultant number from the Doppler shift equation is supposed to be multiplied by 1 (cosine the angle of incidence, that is supposed to be zero Cos 0 =1). If any angulation was faulty made between the U/S beam and the flow, the resultant velocity will be reduced proportional to the cosine of the angle of incidence. **See diagram 13 a & b.**

Diagram 13 A: the effect of cosine angle theta on the velocity.



Notice the typical Ap 4 ch view. With the curser line "U/S beam " aligned parallel to the blood flow from the LA to the LV, the velocity reached 2 m/s.



Notice the angulated Ap 4 ch view. This will lead to an interception angle of about 45 degrees between the U/S Doppler beam and the blood flow from the LA to the LV. This will lead to false reduction in the calculated velocity. The velocity reaches only 1.5 m/s

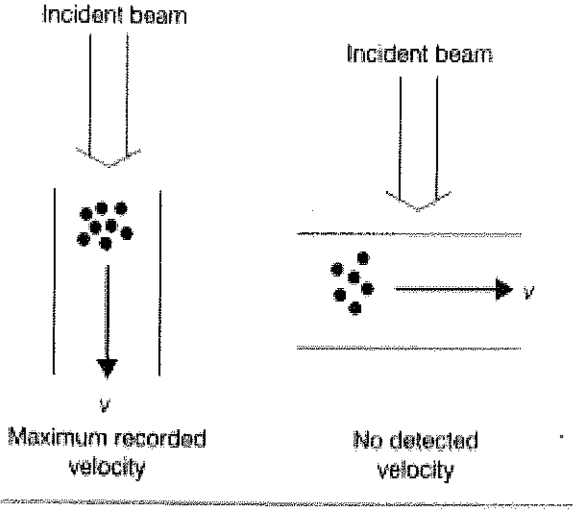


Diagram 13 b: As the angle of incidence between the U/S beam and the flow is zero, the probe will be able to detect the maximal velocity (cosine 0 = 1). With perpendicular angle of incidence between the U/S beam and the flow, the detected velocity will be zero, because cosine 90 = 0.

Pulsed wave Doppler Vs continuous wave Doppler:

Both modalities are used to quantify velocities, however the main difference is that PW Doppler can localize the velocity at a certain point but is subjected to the phenomena of aliasing, so it cannot quantify high velocity jets, while CW Doppler cannot localize the velocity at a certain point but rather measure the highest velocity along the curser line and is not subjected to the phenomenon of aliasing.

Pulsed waves Doppler and the Concept of aliasing in U/S:

Remember from physics discussed on pages 46 -50 that any pulsatile transformer is subjected to the phenomenon of aliasing where the velocities tend to be perceived in an aliased manner i.e. in the opposite direction. Also Remember that what applies to the mechanical wave illustrated from the revolving fan will typically apply to our sound waves.

Now the wave is the sound wave with high frequency, and the probe sends the Doppler U/S in a pulsatile fashion and receives back those waves in a pulsatile fashion too. By the same concept, if the frequency of the U/S waves increases to more than the Nyquist limit, the machine will falsely interpret the velocity as if coming from the opposite direction. See diagram 14 and 17.

Diagram 14

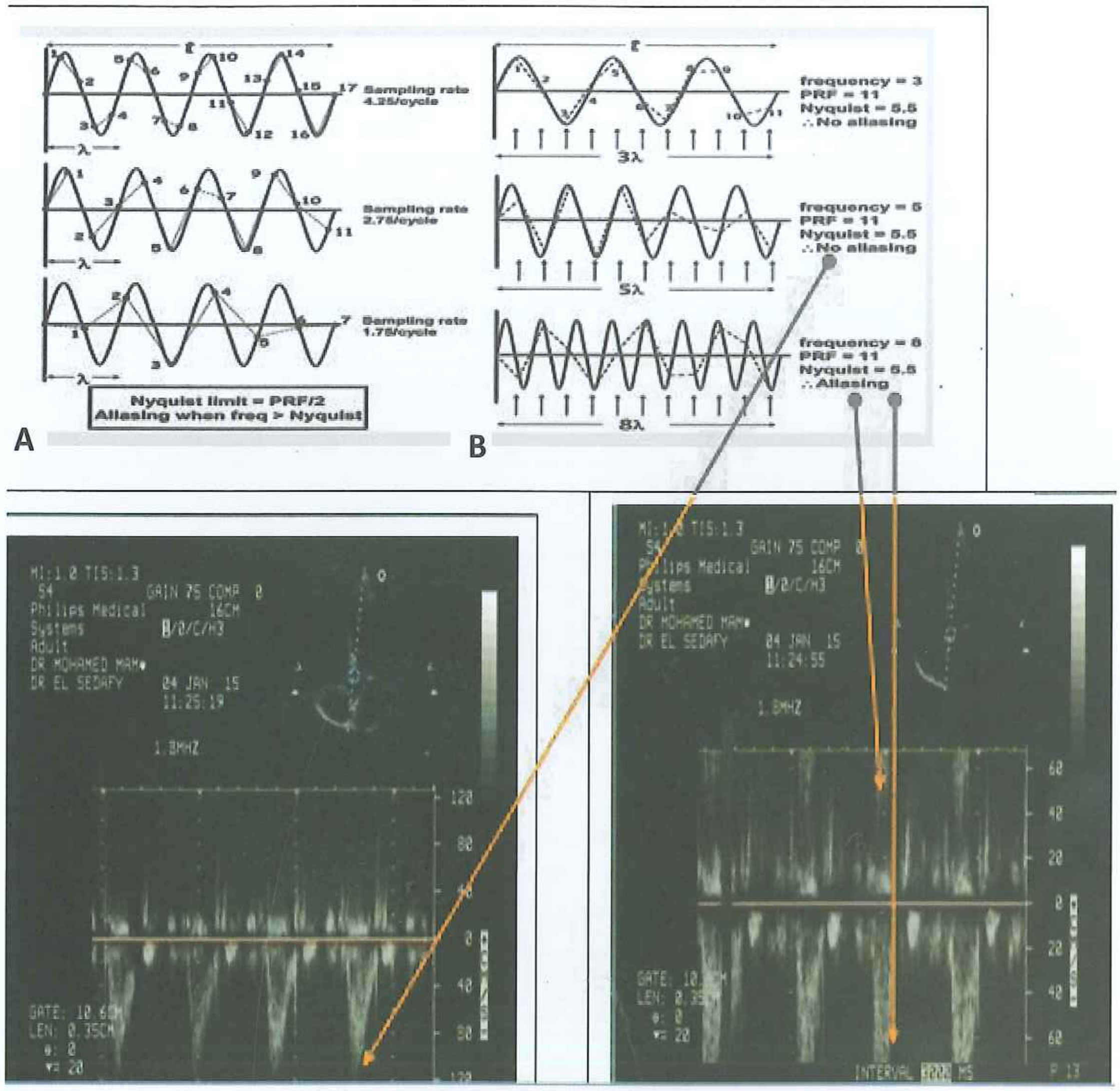


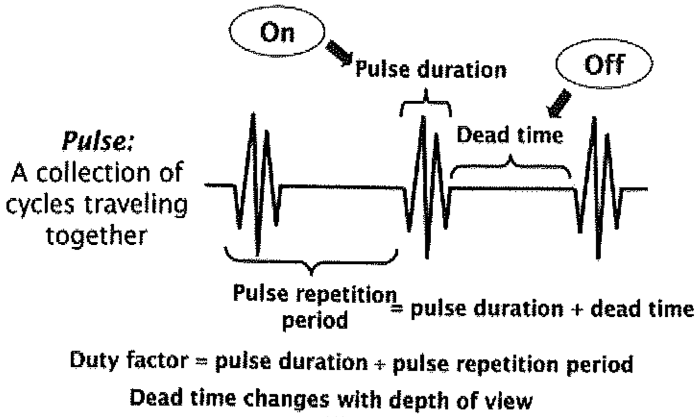
Diagram no 14 : A Doppler study over the aortic folw: ***on the right,*** notice the aliased velocity of the aortic flow (displayed in the opposite direction). This aliasing is due to the fact that the received frequency is higher than the Nyquist limit. Increasing the PRF will eliminate the phenomena.

Aliasing will occur when the F > ½ PRF (Nyquist), which is equivalent to 2F > PRF which is equivalent to PRF< 2 F.

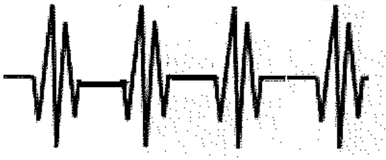
No aliasing will occur when the F ≤ ½ PRF (Nyquist), which equivalent to 2 F ≤ PRF, which is equivalent to PRF≤ 2F.

Notice that the aliased velocity reversed the direction and appeared in the opposite side of the original flow.

So in order to track a high velocity jet the sonographer must increase the PRF (scale) of the machine (by manipulating the PRF button or the Scale button). But this PRF has an upper limit after which the machine must be switched to the continuous wave Doppler. See diagram 15.


On
Pulse duration
Off
Pulse:
A collection of cycles traveling together
Dead time
Pulse repetition period = pulse duration + dead time
Duty factor = pulse duration ÷ pulse repetition period
Dead time changes with depth of view

increasing the PRF lead to shortening in the PRP



with increasing the PRF to certain limit, the PRP will be zero, and the wave will be CW instead of being PW

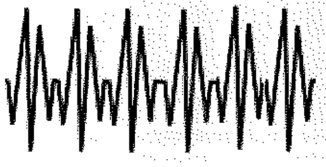
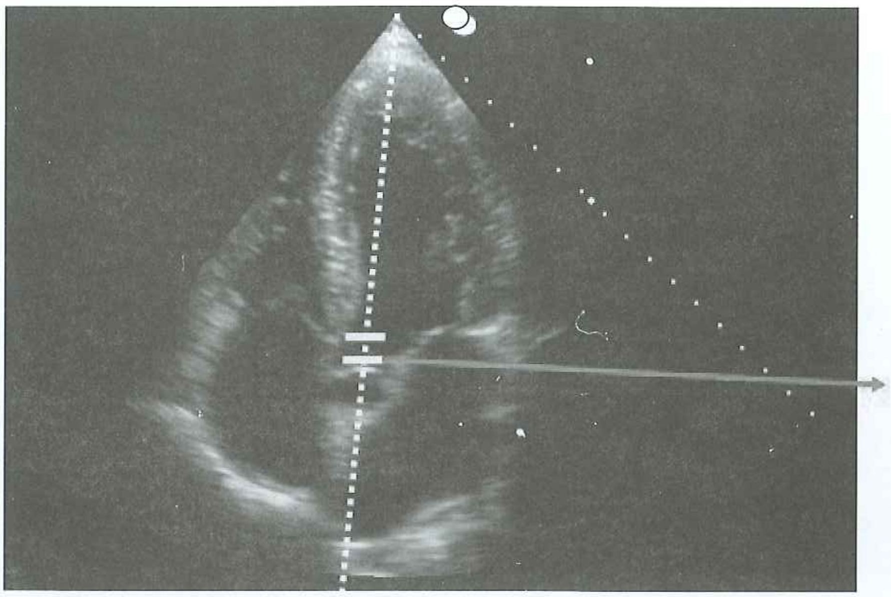


Diagram 15: When the echocardiographer increases the pulse repetition ***frequency*** (PRF), the pulse repetition ***period*** (PRP) will shorten, and at a certain limit the PRP will be zero. At this time the wave is said to be continuous (remember that F=1/P → P=1/F, so by increasing the F, the P decreases. Same concept applies to the PRF and the PRP)

The main advantage in the pulsatile manner is its ability to localize the velocities at a certain point and this is done by the same concept of the range equation. Remember from the 2D imaging is that every dot seen on the screen is understood in terms of time by the machine.

The Speed= distance/ Time, so the Distance = Speed X Time. So if we marked a certain dot we will be actually marking a certain time and then we can easily localize the distance.

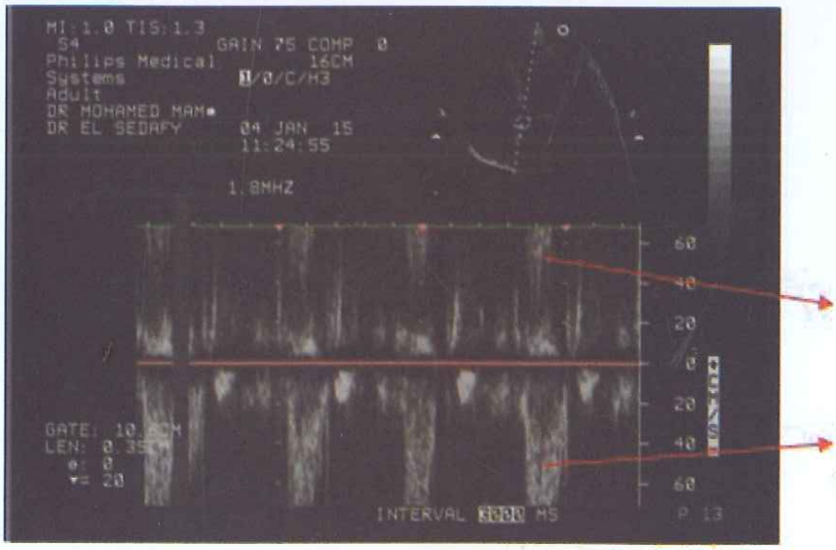
The PW Doppler did not stop at any point but it propagated further than this point and we only controlled the listening time. It is as if we actually told the machine to listen to reflectors received after a certain time and just neglect all other reflectors coming from other distances, so we can only listen from a precise distance. See diagram 16.



Sample volume to determine the exact listing point

Diagram 16 : the sample volume is adjusted in the LVOT, so the machine will only listen to the velocities in the LVOT, neither before nor after.

Diagram 17 (a)

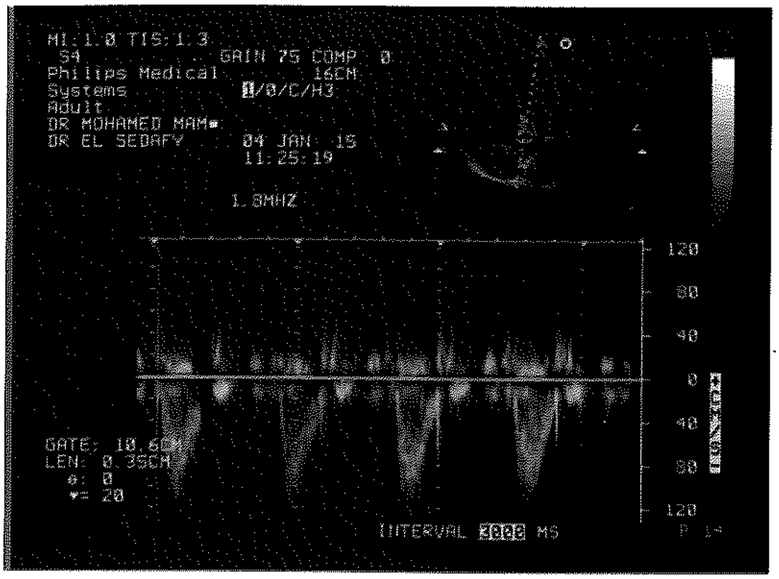


Aliased signal

Original direction of the flow

Aliased signal occurred because the velocity is relatively high so it relatively caused high frequency shifts more than Nyquist limit. To eliminate the phenomenon (aliasing) increase the PRF or the scale.

17 (b)



Increasing the PRF eliminated the phenomenon. (PRF is increased by manipulating the PRF "scale" button).

Continues wave Doppler:

The C.W Doppler is another modality where the probe sends the U/S beam in a continuous, manner, so there will be no dead time (listening time) or blanking out (as what happens with a pulsatile system), thus the probe will be continuously tracking the velocities giving little chance for aliasing to occur, even, if aliasing occurred, increasing the PRF will eliminate the phenomenon. Unlike P.W, C.W has no upper limit for the PRF.

So to track a high velocity jet the sonographer must increase the PRF (see diagram 17). The PRF here has no upper limit, unlike the PW Doppler, and can successfully record very high velocities without aliasing.

As the C.W has no dead time i.e. no listening time, it will not be able to calculate the time interval that elapsed between sending the wave and receiving it. So it will not be able to apply for the range equation, and it will not be able to localize the precise location from where a specific velocity occurred, but it will rather calculate the highest velocity along the examination line (curser) (see aortic stenosis)

Color Doppler:

Another unique modality is the color Doppler. Here the probe sends the pulsatile U/S beam in multiple lines (similar to the 2D echo). BUT the returning signals are analyzed by the Doppler shift equation in terms of color. Again, the probe sends the multiple beam rays in a pulsatile manner with known frequencies and every returning pulse is analyzed by the Doppler shift equation into velocities (i.e. speed and direction) inside the computer's memory. These velocities are translated into so many graphs (the same graphs of the velocities discussed before). Every sent and received pulse correlates to a graph inside the computer's memory. Each graph is color encoded according to its Doppler shift, typically into red, blue or mosaic dot. Most commonly the positive Doppler shifts are encoded into red dots and the negative Doppler shifts are encoded to blue dots. The mosaic color encodes for the aliased velocity. ***For***

more clarification, the blood flowing towards the probe will be in red color and the blood flowing away from the probe will be in blue. So, Jets with normal speed will be encoded either into red or blue according to the direction, and the high speed jets will be typically encoded with a blue mosaic or red mosaic (see diagram 18 a & b). This mosaic pattern corresponds to the turbulence that occurs in any high velocity jet.

It is crucial to remember that the color Doppler encodes the blood flow in terms of velocity, i.e. direction and speed, so low speed +ve Doppler shifts will be encoded in red, high speed +ve Doppler shift will be encoded in mosaic red, low speed –ve Doppler shifts will be encoded in blue, and high speed –ve Doppler shifts will be encoded in mosaic blue. (High speed = mosaic)

N.B: Each scan line is formed of 10 pulses

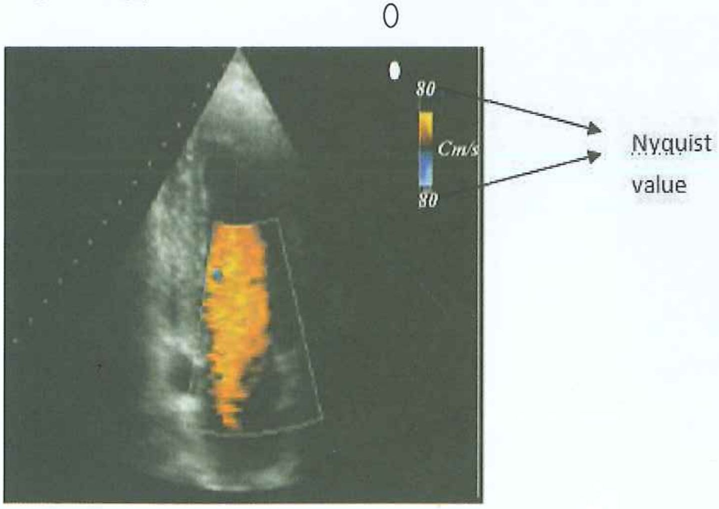
The mosaic appearance of the color Doppler is referred to as aliasing.

Aliasing means that the velocity has increased more that the set Nyquist limit (i.e. the frequency shifts are more than the ½ PRF value leading to aliasing).

When setting the echocardiography machine on the color Doppler (by pressing the color Doppler button), a color map appears at the side of the screen with two values at both ends of the scale. See diagram 18a. Those numbers are referred to as the Nyquist limit (a physics scientist). They are equivalent to the half value of PRF (if using PW Doppler), I.e. aliasing limit. **Any velocity that will exceed this value (speed) will be encoded in a mosaic pattern.**

Diagram 18 (a)

Diagram 18 (a)



Normal mitral flow using color Doppler, notice the red color denoting a normal low velocity jet.

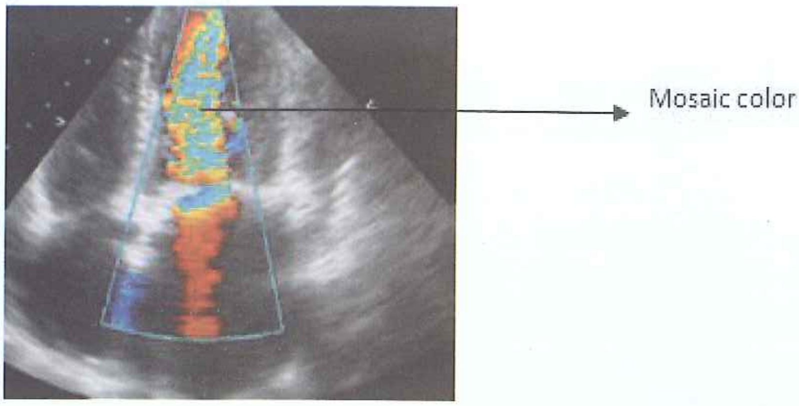
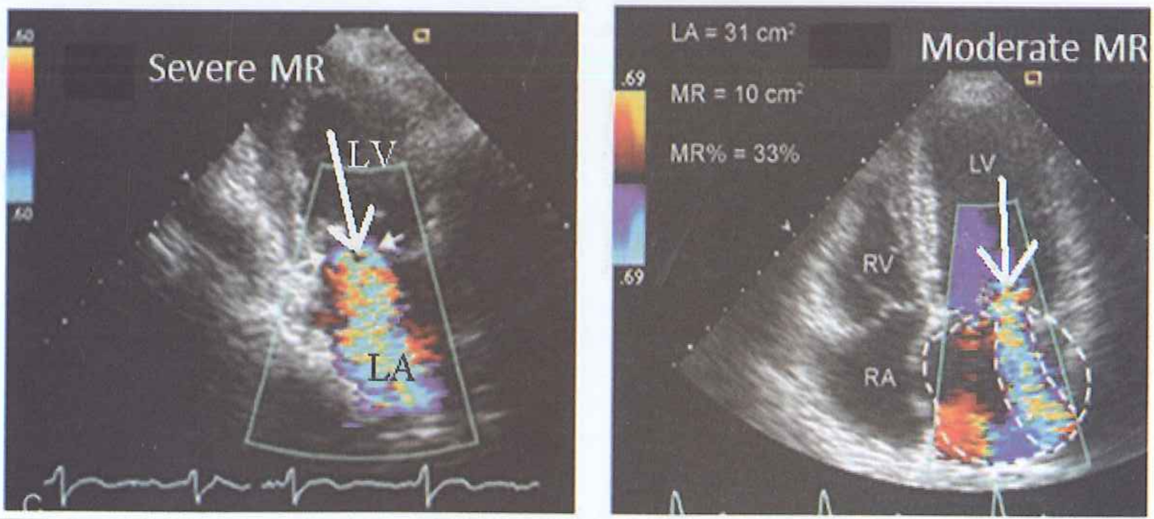


Diagram 18 (B): Transmitral high velocity jet flow is encoded in red mosaic color.

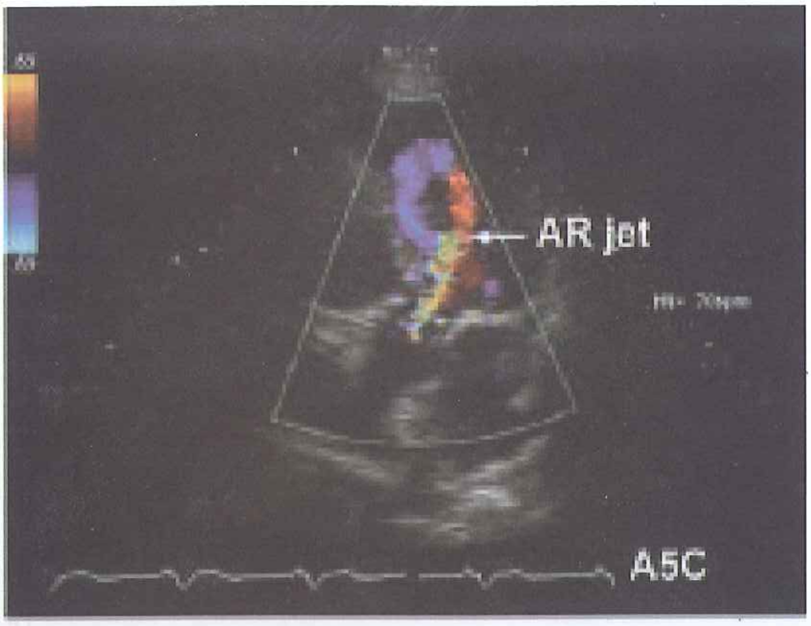
N.B: Remember that the Nyquist limit is the half value of the PRF (See before).

So when setting the machine at high PRF (high Nyquist limit) this means that the sonographer has set the machine to read relatively high velocity jet in a non mosaic pattern, and setting the machine at a low PRF means that the sonographer has set the machine to read relatively low velocity jet in a mosaic pattern. I.e. with high PRF settings, the flow must reach high velocity to be encoded as mosaic, while with low PRF any minimal increase in the velocity of the jet will be make the flow to be encoded as mosaic. Example: when detecting a low velocity jet as in an ASD, the sonographer has to decrease the PRF in order to increase the sensitivity of the machine to detect the low velocity left to right shunt. While during tachycardia the sonographer may need to increase the PRF in order to compensate for the increased velocities and reduce the amounts of mosaic appearance.



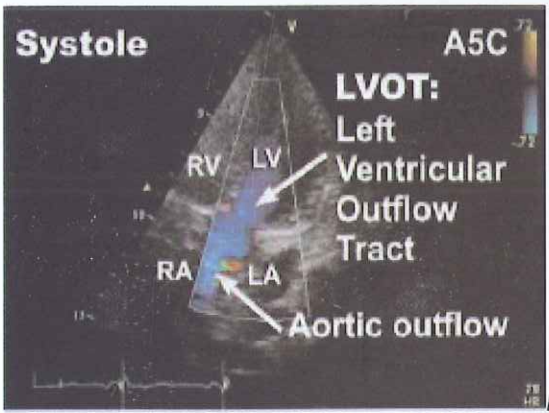
Notice the blue mosaic pattern regurgitating from the LV to the LA, (denoted by the white arrow), so mitral regurgitation will lead to a negative Doppler shift and will be encoded in blue color, but due to the high PG between the LV and the LA during systole, MR is encoded in red mosaic (see text for details)

Normally no blue mosaic color should be elicited in the LA.



Notice the red mosaic pattern regurgitating from the aorta to the LV, so aortic regurgitation will lead to a positive Doppler shift and will be encoded in red color, but due to the high PG between the aorta and the LV during diastole, AR is encoded in red mosaic (see text for details)

Normally during diastole no red mosaic color should be elicited filling the LV.



Normal color flow of the LVOT during the systole.

Pitfalls in color Doppler :

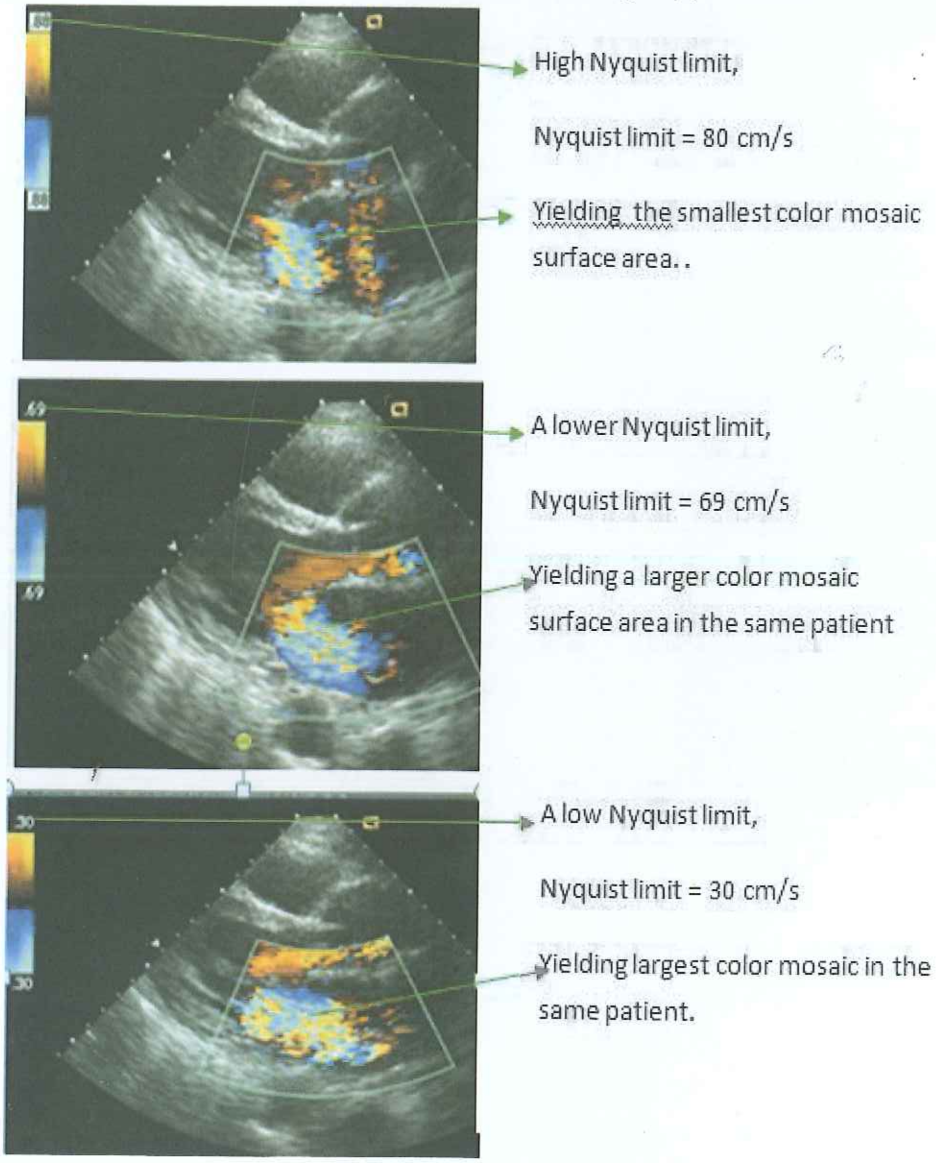
1) **Any factor that causes a reduction of the jet speed will underestimate the amount of the mosaic jet and vice versa**. See mitral regurgitation.

2) **Different PRF settings will lead to different results,**

Example 1: if the two different sonographers performed echocardiography examination for a patient with mitral regurgitation using different PRF values, the one using a lower PRF values may have a tendency to overestimate the MR, while the one using a higher PRF values may have a tendency to under estimate the severity of the MR. see diagram 19.

I.e. with a high PRF, a severe regurgitation lesion may appear moderate, and with a low PRF a mild regurgitation lesion may appear moderate by color Doppler.

Diagram 19 : showing the effect of decreasing Nyqiust limits on the color jet surface area



High Nyquist limit,

Nyquist limit = 80 cm/s

Yielding the smallest color mosaic surface area. .

A lower Nyquist limit,

Nyquist limit = 69 cm/s

Yielding a larger color mosaic surface area in the same patient

A low Nyquist limit,

Nyquist limit = 30 cm/s

Yielding largest color mosaic in the same patient.

N.B: if an ASD is examined with a relatively high PRF the left to right flow may be completely over looked.

3) Gain settings:

Gain is defined as the amplification of the received signal.

Sometimes the sonographer has to increase the gain settings of an echo modality in order to compensate for the poorly received signal.

Over calibration of the gain settings while using the color Doppler will lead to an increase in the mosaic pattern and vice versa (the net result will be like manipulating the PRF, but the PRF is a preprocessing parameter while the Gain settings is a post processing one)

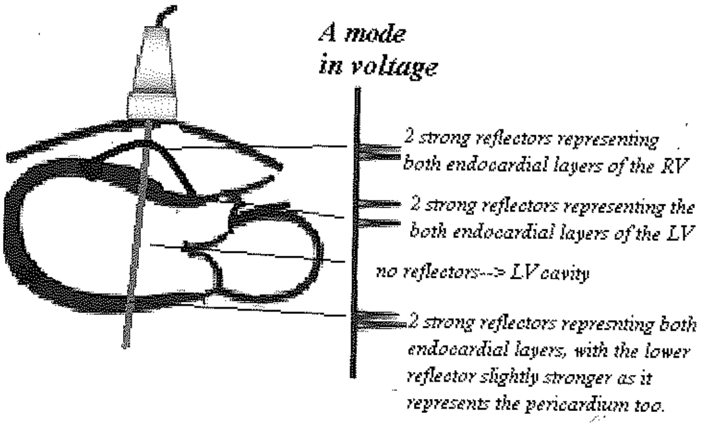
3) Different machines:

They use different encoding systems to encode for the flow. This leads to a slight variation in the results when using different machines to judge the severity of a lesion using only color Doppler.

Chapter 4: Echocardiography modalities.

The, Amplitude mode "A mode":

The first modality in echocardiography depended on sending the U/S beam in a single line from only one piezoelectric crystal and then receiving back the reflected U/S beam from various structures "various reflectors". See diagram 20.


A mode
in voltage
2 strong reflectors representing both endocardial layers of the RV
2 strong reflectors representing the both endocardial layers of the LV
no reflectors--> LV cavity
2 strong reflectors represnting both endocardial layers, with the lower reflector slightly stronger as it represents the pericardium too.

Amplitude mode. A mode

Diagram no 20

The data received was displayed in voltages in a way that the strong reflectors as endocardium and pericardium will reflect the U/S beam stronger than weaker reflectors as the myocardium. These strong reflectors will cause better conformational changes in the piezoelectric crystals that will turn those reflected waves back to relatively higher voltages than those from weaker reflectors. (The concept of piezoelectricity, see before)

So strong reflectors will lead to higher amplitude voltages, and weak reflectors will lead to lower amplitude voltage . See diagram 20.

N.B the A mode applies the same concept of the range equation but with a different displaying process owing to the different technology.

Brightness mode “B mode”:

For sure the A mode was so difficult to be analyzed by Doctors. Later on, scientists displayed these voltages in a more understandable manner in a way that high voltages will be displayed in very bright white dots and lower voltages will be displayed in less brighter dots, what's called “the grey scale". For example, the posterior wall appeared as 2 bright white dots, with grey dots in between, the two bright dots corresponds to the endocardium and the pericardium "strong reflectors" the grey dots corresponds to the myocardium "weaker reflector". In some situations, the myocardium may not be displayed at all, leading to what is known as dropping out. (causes of the dropping out are discussed later). Until that time doctors were able to see the walls of the heart only in an axial orientation without any lateral orientation. See diagram 21.

N.B : The B.mode applies for the same concept of the range equation but in a different display mode due to a different technology .

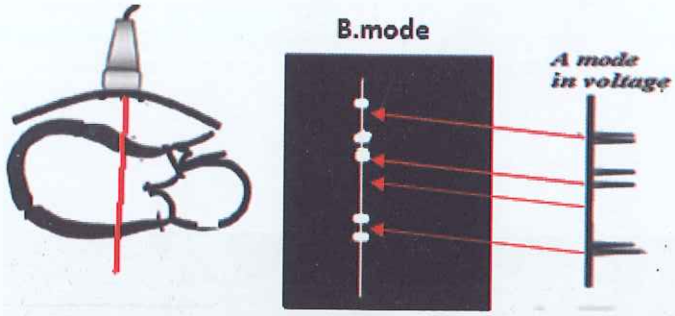


Diagram no : 21

Motion mode "M.mode":

By convenience, these bright dots gave very little information for the cardiac dimensions, because all the sent and received information were received without respect to time , to understand this , imagine an ECG (Electrocardiogram) with needle going up and down without the paper being rolled out !. It will draw overlapping lines and all waves will be displayed as lines at the same place. But with the paper being rolled out the waves will be stretched in respect to time drawing the well known P QRS T waves. The same concept explains the difference between the brightness mode and motion mode (M.mode). See diagram 22.

Remember that the myocardium is a weaker reflector than both the endocardium and the pericardium, and normally the cardiac wall in the "M.MODE." will appear as two bright lines with hypo echoic area in between.

N.B the M.mode applies the same concept of the range equation. (remember that M.mode is a modification of the B.mode)

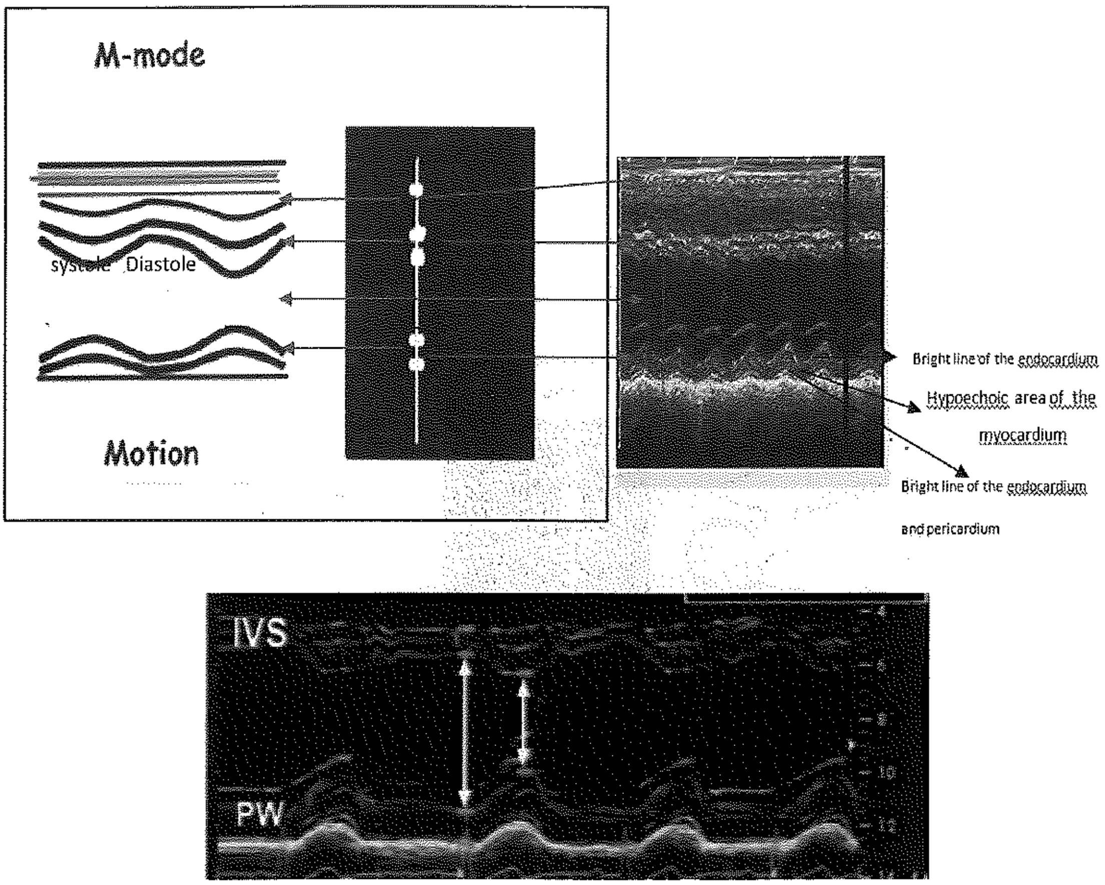
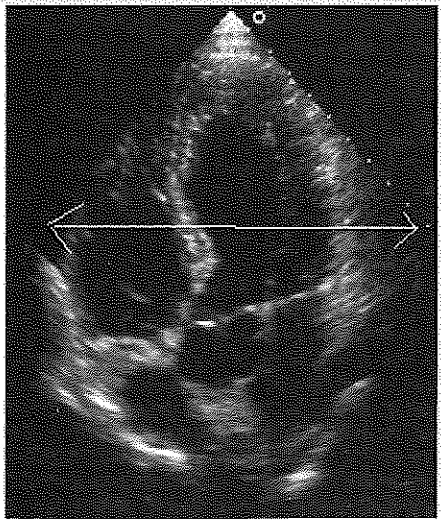

M-mode
systole
Diastole
Motion
Bright line of the endocardium
Hypoechoic area of the myocardium
Bright line of the endocardium and pericardium
IVS
PW

Diagram 22 : Notice that the cardiac wall normally appears as two bright lines with a hypodense echogenic area inbetween, i.e. a dropping out (myocardium) is normally seen in-between the two bright lines of the pericardium and the endocardium.

Anatomical M.mode "M.mode steer" :

A soft ware technology that enables the sonographer to simulate the M.mode alignment but in any needed angle. See diagram 23

Diagram 23 for anatomical M.mode.



the M.mode cursor can be manipulated freely, the M.mode cursor can be manipulated to cut laterally as shown by the arrow.

N.B: No U/S beam actually can be steered in such a way as shown in the diagram, but the soft ware of the machine stretches the required points; indentified by the steered curser; over time.

2 Dimensional imaging "2D":

As a natural evolution, instead of using just one piezoelectric crystal , Scientists used many piezoelectric crystals aligned in a line within the probe, thus forming many lines of the U.S beam see diagram 24, each line will represent a bright dot, so, many bright dots adjacent to each other will form the 2 D image. Now we can see both the axial orientation and the lateral one. Each impulse sent and received by the probe will represent and form one frame. Sending and receiving successive impulses will form many frames. Playing these frames one after another will create a 2 D video. (Playing these 2D images against time will produce the real time 2 D videos.)

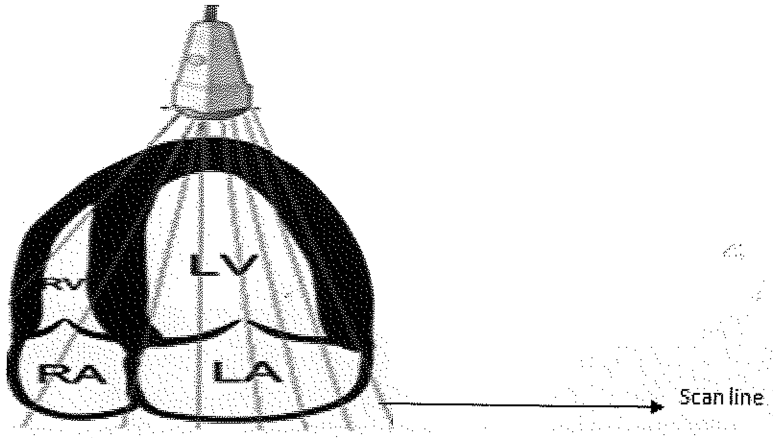


Diagram 24: showing multiple scan lines, each scan line is formed by a single piezoelectric crystal.

Important and mandatory considerations on acquisition of the 2D image :

1) Optimizing the gain settings : "gain button" / TGC buttons
   N.B : the word "gain" is used as an equivalent to the word "increase"

Remember from the "A" and "B" modes that the different cardiac layers are displayed in terms of the power of the reflected U/S waves, so, in some situations the U/S waves hits a poor reflector so the reflected U/S waves will be scattered, and the amount of the U/S waves received by the probe is weak and thus generates weak conformational changes in the piezoelectric crystals. This will lead to a low voltage display and subsequently hypo echoic area "dropping out". This "dropping out " area must be identified by the sonographer in order to identify the true cardiac wall. One of the important settings that can help to overcome this phenomena is the "Gain settings". **Gain is the amplification of the received signals (a post processing parameter).** By increasing the gain settings , the low voltages; that was generated due to the weak U/S reflections; will be increased (amplified), this will cause a marked discrepancy between the true cavity (i.e. no reflectors & no voltages generated) and the actual cardiac dropped out area, i.e. the dropped out area will appear as a true wall with much increased echogenicity. See diagram 25 (A and B).

Diagram 25 A: Effects of the high gain settings on the 2 D image. Notice the poor image quality using excessively high gain settings.

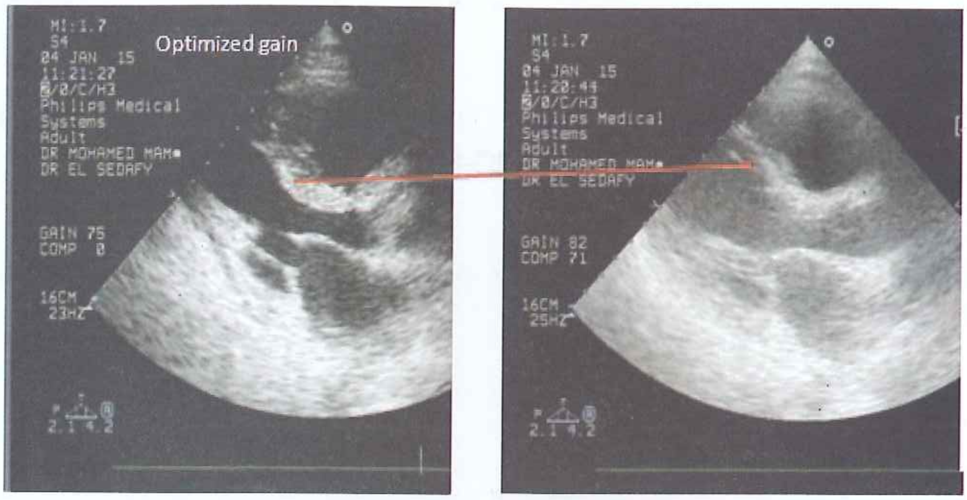
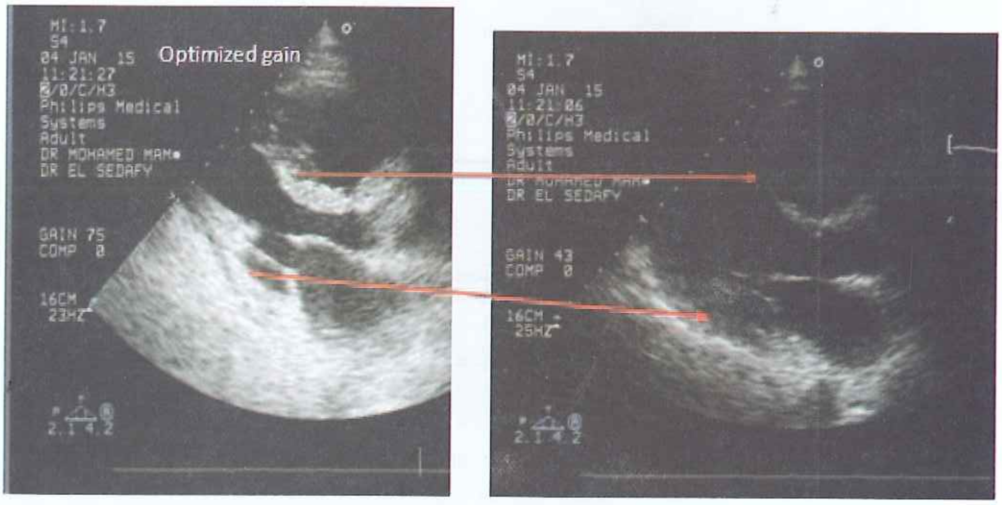
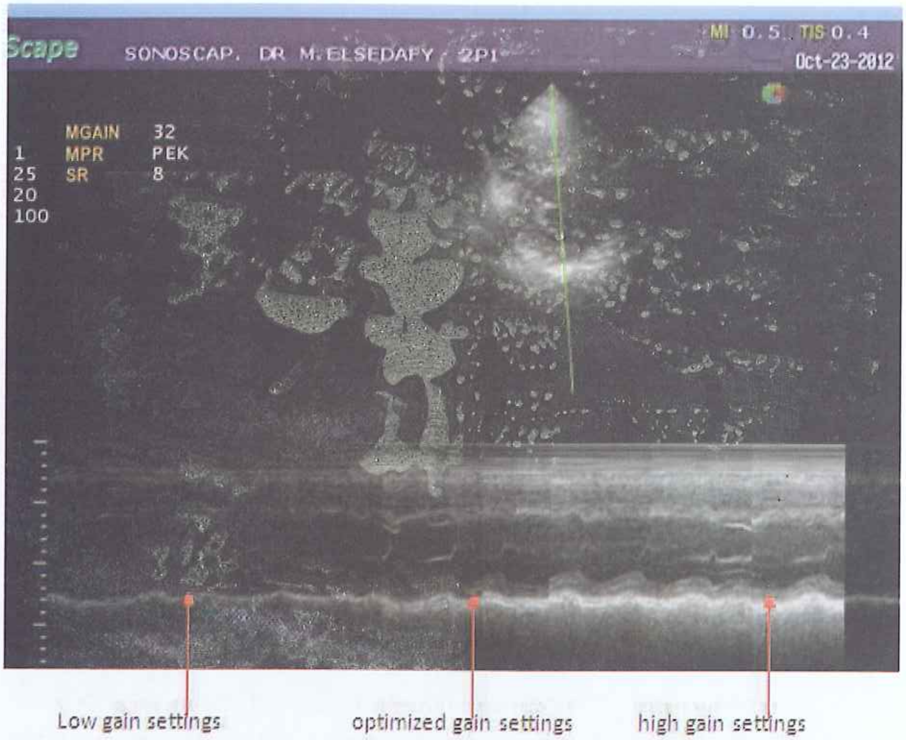


Diagram 25 B : effect of low gain settings on the picture. Notice the dropping out of the septum and the posterior walls.



Failure to adjust the gain parameters will lead to hypo echoic areas and even a total dropping out of the endocardial layer (complete loss of visualization) leading to faulty measures of the intracardiac dimensions. See diagram 26

Diagram 26 : M.mode trace showing the effect of the different gain shade grades. (N.B patient had A.F and paradoxical septal motion which will be discussed later)

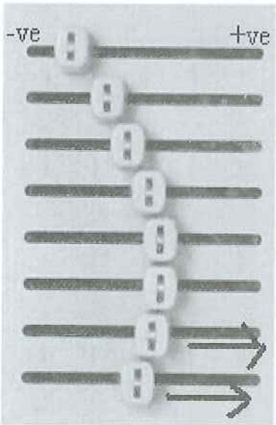


With low gain, notice the dropped out posterior myocardial and endocardial layers, leaving only the epicardial and the pericardial walls. Measuring with low gain settings will lead to over estimation of left ventricular diastolic dimensions.

Causes of poor U/S reflections during 2 D imaging:

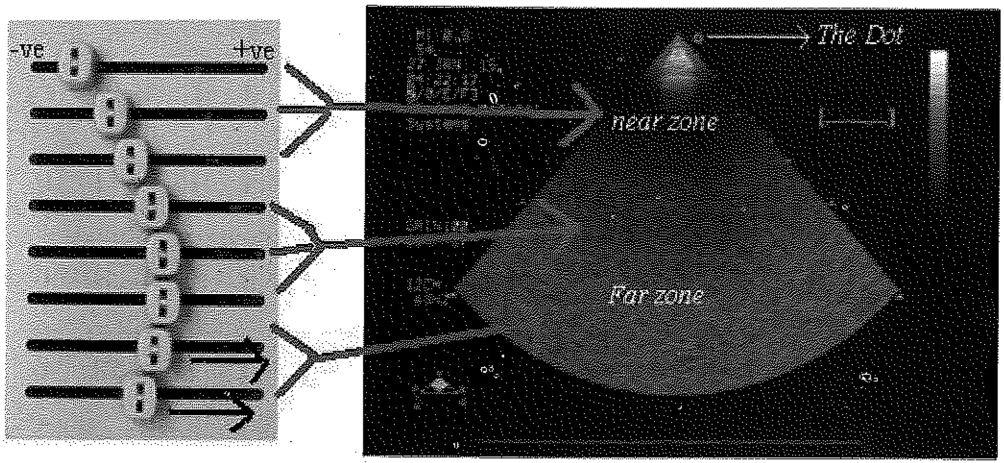
A) oblique angle of incidence between the U/S beam and the tissues.

B) small reflectors in relation to the wave length of the U/S beam.



The Time Gain Compensation buttons "TGC" : these are typically equivalent to the gain button, but can perform the amplification of the received signals returning from different depths **selectively.** i.e. : in an apical 4 chamber view the apex is a near reflector and the base is a far reflector (the base lies in the far zone), so the reflected U/S beam from the base has to travel longer distances than those returning from the apex, so the U/S beams returning from the base will have lost more energy during their longer journey. This means that the U/S beams returning from the base will be weaker than those reflected from the apex and will cause weaker conformational changes in the piezoelectric crystals that in turn will be displayed by weaker voltages and decreased echogenicity. By shifting the downward two buttons in the TGC towards the +ve side, the sonographer will perform amplification of the in the U/S beam returning from the far zone, **so we will be able to compensate and increase the U/S**

beam signal reflected from the further reflectors for their longer time journey, and hence the name Time Gain Compensation.



Adjusting the power of the U/S beam.

This is done by calibrating the "power" button either to increase or decrease the transmitted power (preprocessing parameter). By increasing power, the machine delivers higher voltage to the piezoelectric crystals, leading to stronger vibrations (increased amplitude in the vibrations) within the piezoelectric crystal leading to increased amplitude of the transmitted U/S beam, (remember this is totally different from the frequency and wave length) (see page 42 & 43). the relationship between the voltage given by the machine and the resultant power is displayed in this equation : $dB = 10 \log P_2/P_1$.

So the power is directly proportional to the Amplitude^2.

So it is logical that the sonographer has to increase the power of the machine in conditions like obese patient, thick chest wall, poor echogenicity. Most schools suggest that the first parameter to adjust in the condition of poorly echogenic picture is the Power. However the **EMSE** suggests that the sonographer has to operate with the least possible power that gives a convenient picture. (increasing the power decreases the lifespan of the piezoelectric crystals). This tip is very important in a transesophageal study where increasing the power will over heat the transducer and may lead to a temporally cut off of the picture until it cools down again.

N.B: Failure to adjust either the power or gain settings will lead to the same final effect on the picture acquisition.

N.B: Beam intensity (W/cm^2) = power (Watts) / beam area (cm^2)

The beam intensity measures how much power is concentrated in 1 cm^2.

2) Adjusting the frequency of the U/S beam according to the clinical situation

This is done from the frequency button or by changing the probe.

Changing the frequency affects both the penetration and the resolution. See diagram 27.

Remember from clinically applied physics that the speed of U/S (C) = wave length (λ) X frequency (F), and that the speed of the sound wave is constant according to the medium. C=λ X F

So, any increase in the frequency will lead to a directly proportional decrease in the wave length to keep the speed constant (a physical fact) and vice versa.

Increasing the frequency will cause more friction between the U/S and the medium (with high frequency the wave repeats itself many time within a given medium leading to increased friction), this will lead to increased resistance and attenuation in the wave intensity and thus a decreased penetration. With low frequencies the opposite occurs.

So, in obese patients it is better to decrease the frequency in order to increase the penetration of the U/S beam and thus optimizes the image quality.

The main problem in decreasing the frequency is the resultant larger wave length.

The larger the wave length the lower the resolution of the picture.

So in a pediatric case, we do not need much penetration (same idea with linear probes and superficial examination i.e. thyroid U/S exam) and it is convenient to use a probe with high frequency (for example 8 MHz) that will give a better resolution of the small structures.

The tradeoffs between penetration and resolution: increasing the penetration will decrease the resolution, and decreasing the penetration will increase the resolution.

We can only control the frequency of the U/S wave. This is done by changing the thickness of the piezoelectric crystals and the bandwidth. The wave length changes accordingly.

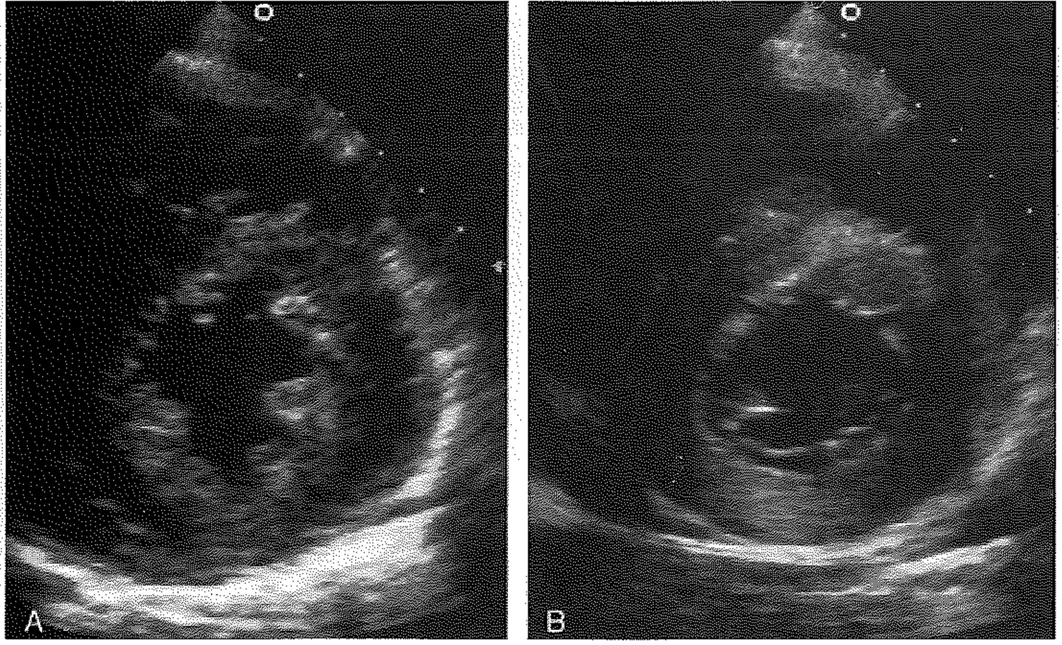


Diagram 27: The effects of different transducer frequencies on image quality and appearance are demonstrated; A: A 3.0-MHz transducer is used to record a short-axis view, B: The same image is recorded using a 5.0-MHz transducer.

Attenuation coefficient: the factor by which the power of the U/S beam is reduced by for every cm traveled by the U/S beam.

Attenuation coefficient = ½ F

Total attenuation = path length X attenuation coefficient

Penetration depth = 3/ attenuation coefficient. (The penetration depth is the depth at which the intensity is reduced to one half its original value)

Impedance (resistance) = density (Kg/m^3) X propagation speed (m/s)

F= the material propagation speed/ double the thickness of the material. (i.e. as the thickness increases the F decreases)

High frequency U/S has narrower beams and diverges less than wider beams.

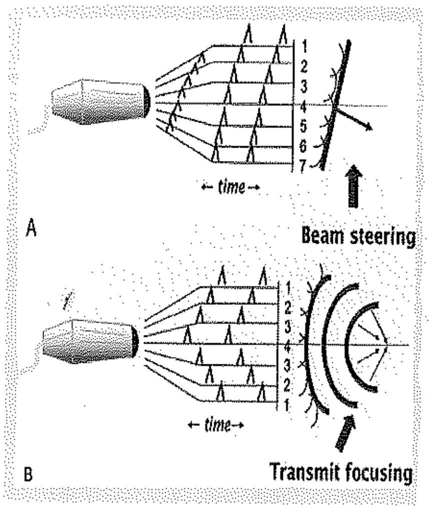
N.B: the frequency of the Echocardiography probe ranges from 3 to 12 mega Hz. (adult: 3-5 MHz, pediatrics: 8 MHz, neonates: 12 MHz)

3) Optimizing the focus to the desired area:

This is done by adjusting the "focus" button. By adjusting the marker (as shown diagram 28) on a certain area, the U/S beam is focused on this area of interest, thus they reflect best from this area and enhance the image quality.

This is the same concept of focusing light by a lens.

Previously, this was done using an acoustic lens. In recent technology, this is done by beam steering/ focusing using the concept of sequential firing. See diagram 28.



Phased-array technology permits steering of the ultrasound beam. By adjusting the timing of excitation of the individual piezoelectric crystals, the wave front of ultrasound energy can be directed, as shown. Beam steering is a fundamental feature of how two-dimensional images are created. B: By adjusting the timing of excitation of the individual crystals within a phased-array transducer, the beam can be focused. In this example, the outer elements are fired first, followed sequentially by the more central elements. Because the speed of sound is fixed, this manipulation in the timing of excitation results in a wave front that is curved and focused. This is called transmit focusing.

Diagram 28 : beam focusing / steering.

So, to enhance the image quality at a certain point, simply adjust marker of the focus at its level. See diagram 29.

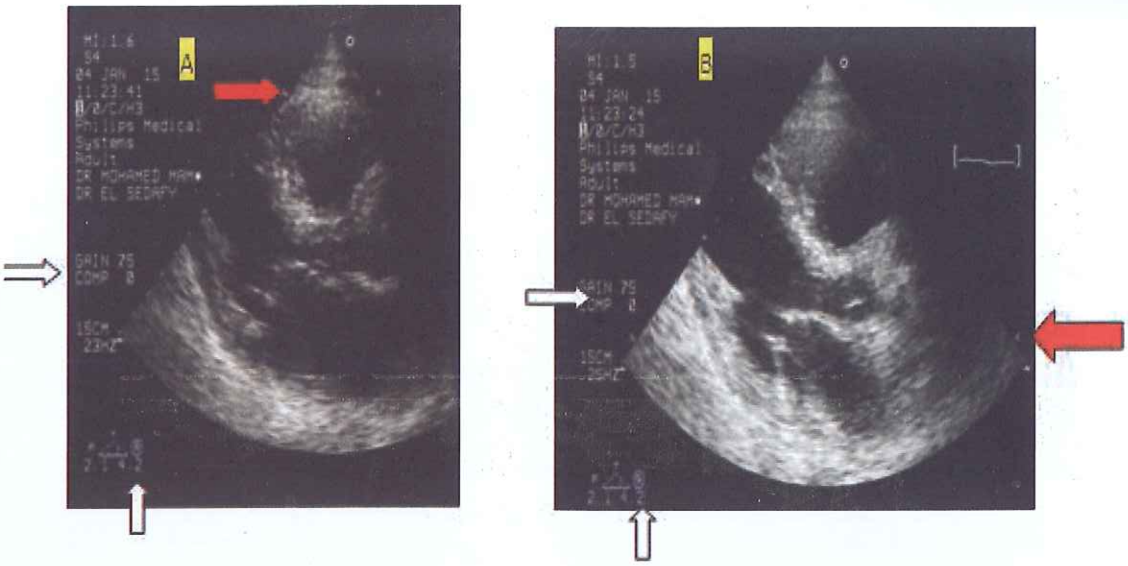


Diagram 29 : notice the same gain , same frequency (shown by white arrows), see the effect of the different focusing on the clarity of the mitral valve (shown by red arrows) .

A : poor focusing over the M.V B : optimized focusing at the level of the M.V.

N.B: focal length = (transducer's diameter ^{2} X F) / 6

The U/S beam produced by the small-diameter crystals diverges more than U/S beam produced by large diameter crystals and vice versa.

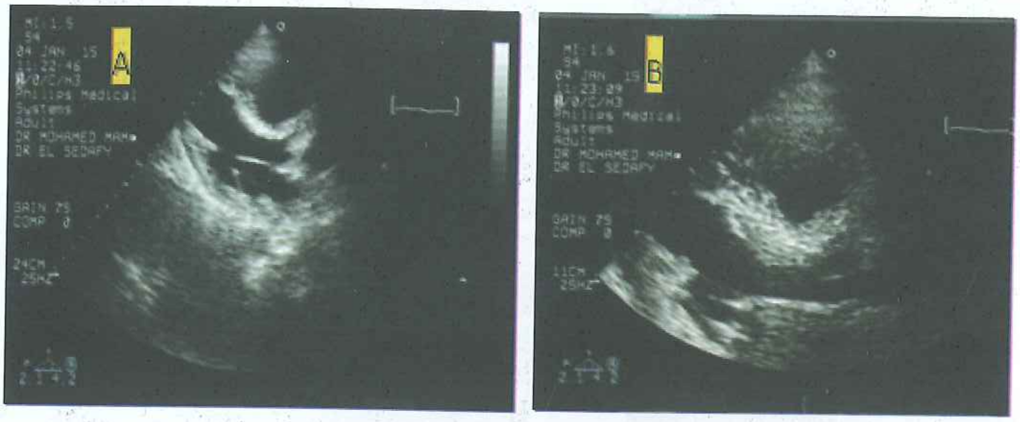
4) Adjusting the depth:

This is done by adjusting the "Depth" button.

By this bottom the sonographer can select to image either deep structures or more superficial ones. See diagram 30 a & b & c .

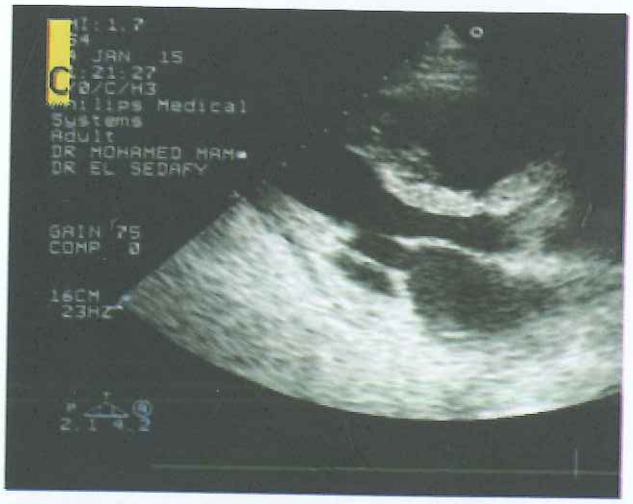
Increased depth as deep as 24 cm will give inconvenient images.

Diagram 30



Increased depth as deep as 24 cm will give Inconvenient images.

Superficial imaging depth to only 11 cm will lead to a failure in imaging of the LA



Optimized depth will give the best image quality (best temporal resolution)

N.B :

*The main problem for a beginner sonographer is the shift from a PLAX view to an apical view. A lot of beginners forget to adjust the depth. See diagram

*Taking the measurements with a suitable depth increases its accuracy. See example in diagram.

By increasing the depth, the time of the flight made by the U/S wave will be increased. So the manufacturer must increase the pulse repetition period to allow for more listening time to give "time chance" for the far reflectors to return back to the piezoelectrical crystal and "catch up" the piezoelectric crystal while it is still in the listening time in order to be displayed. If the far reflectors did not "catch up" the listing time, this means that it had returned during the firing of the next pulse (i.e. the piezoelectric crystal is vibrating and cannot generate voltage) and so the returning impulse will not be displayed. This will lead to marked image distortion.

So by increasing the depth the sonographer actually increased the PRP and decreased the PRF. Remember that for each pulse generated and captured back, one image frame is acquired. So by decreasing the rate of impulse firing (↓ PRF) the number of frames acquired per second is decreased which means a reduction in the image temporal resolution (temporal resolution refers to the number of frames per second. If the number of frames per second is low, the playing video will appear stuttering)

PRF= 1/PRP, therefore the PRP = 1/PRF , duty factor = pulse duration / PRP (see diagram 9)

Therefore the duty factor = pulse duration X PR. So by increasing the depth the PRF decreases and the duty factor decreases too.

5) Adjusting the Sector plane:

This is done by selecting the option of "sector plan adjustment" then moving the track ball. By decreasing the sector plan, the U/S beam line intensity increase. This enhances the image quality and increases the resolution.

N.B reducing the sector plan increases the temporal and lateral resolution.

For example : if it is difficult to judge an aortic valve in a PSAX whether it is bicuspid of tri-leaflet valve, reducing the "sector plan" to include only the aortic valve, may delineate the cusps more clearly during opening and closing. That was achieved due to a better temporal resolution.

Spatial resolution is the ability of the machine to display structures that are situated close together as separate entities.
It is divided into lateral and axial resolution.
Lateral resolution : refers to the ability to differentiate between two points that lies side by side horizontally and depends on:

1) the number of scan lines in a sector (the more scan line the better the lateral resolution)
2) the width of the beam (improves with focusing)(the thinner the better)
3) the amount of averaging or smoothing that the machine applies.
   Typical lateral resolution is 1-3 mm.

Axial resolution :is the ability of the machine to differentiate between two points that lies behind each other i.e. on the same axis along the length of the beam, and depends on :

1) the length (in distance) of the ultrasound pulse (pulse duration) .
2) It tends to be more accurate than lateral resolution, (0.5-2 mm) which is why distance measurements are more accurate vertically than horizontally. The best application of this principle, therefore, is in measurements made on M-mode traces.

N.B :The higher the frequency of the transducer, the better the axial and lateral resolution. Higher frequencies give shorter pulse lengths, and smaller crystals give rise to narrower beams.

Temporal resolution: resolution in relation to time, i.e. the ability to differentiate points during a playing video.

Imaging fast-moving structures, such as the heart, requires a high frame rate so that continuous movement is seen by the eye and there is no obvious jerkiness from one frame to the next. An acceptable rate is 20-40 frames/s (each frame is acquired by an impulse) . A fast heart rate will require a higher frame rate for equivalent diagnostic information to be obtained (e.g. if the frame rate is 30 Hz, an adult heart rate of 60 bpm will mean 30 images of every cardiac cycle, but in a neonate with a heart rate of 150 bpm there will only be 12 images of every cardiac cycle).

Temporal resolution depends upon:

1) Pulses per scan line, so multi focusing decreases the pulses per scan line and decreases the temporal resolution.
2) Sector size and lines per angle of the sector, so by decreasing the sector plane the line density increases and the temporal resolution increases.
3) Imaging depth, with far imaging the PRF must decrease to allow for more listening time to perceive the reflected U/S beams from the deep structures, slower PRF will lead to less frames per second. So increasing the imaging depth decreases the temporal resolution.

REFLECTION AND TRANSMISSION

INCIDENT INTENSITY : the intensity that the sound wave possesses at the instant prior to striking a boundary.

REFLECTED INTENSITY : the intensity that, after striking a boundary, changes direction and returns back in the direction it came from.

TRANSMITTED INTENSITY : the intensity that, after striking a boundary, continues on in the same general direction that it was traveling.

Units: W / cm^2 (*they are all intensities!*)

incident (starting) intensity = reflected intensity + transmitted intensity

N.B :in clinical imaging, very little of the US intensity is reflected back to the transducer at a boundary between soft tissues. The remainder is transmitted and continues to propagate in the forward direction.

INTENSITY REFLECTION COEFFICIENT (IRC) : is the percentage of the US: intensity that is bounced back when the sound beam passes from one medium to another. i.e. these are the intensities of the U/S beam that are reflected back & hits the probe to be utilized by the echocardiography machine.

Intensity transmission coefficient = transmitted intensity / incident intensity

= 1- intensity reflection coefficient

incident intensity (W/cm^2) = reflected intensity + transmitted intensity

transmitted intensity = incident intensity x intensity transmission coefficient

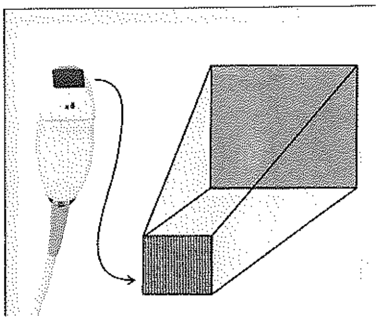
IRC % = { impedance 2 – impedance 1 / impedance 2 + impedance 1$\}^2$

i.e. U/S wave only reflects when it intercepts a different medium with different impedance.

3 Dimensional imaging "3D imaging" :

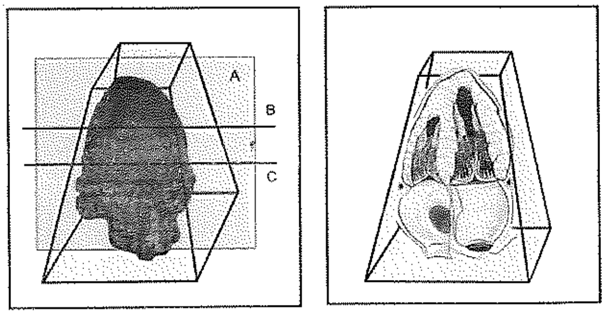
Instead of aligning the piezoelectric crystals in one straight line within the probe, manufacturers aligned them in many straight lines , so instead of forming one plane of U/S wave many planes can be generated forming a cone like projection of the U/S beam (see diagram 31) , for sure this cone like projection will reflect back from the heart forming a 3 dimensional image of the heart. Then the sonographer can selectively slice the heart in any projection seems to be rationale.

diagram 31



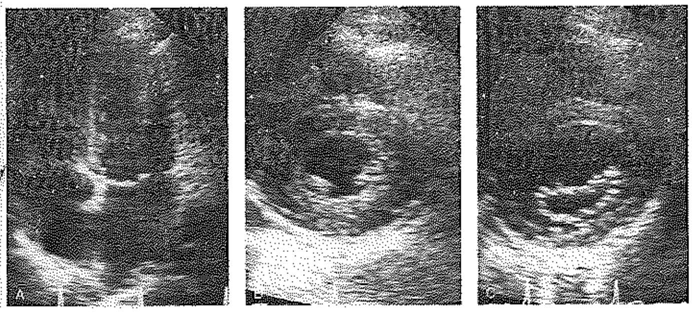
A three-dimensional volumetric probe. Note the relatively square footprint of the probe compared with a standard two-dimensional probe. The face of the probe consists of a nearly rectangular matrix of crystals that results in a pyramidal scan profile, as depicted in the schematic on the right.

Diagram 32 : illustrating the principle of the 3 D echo:



Unprocessed Full Volume

"Cropped" Full Volume Data Set



3 D echo gives the utility for unlimited cur sections in a three dimensional

Summary of 2 D imaging :

The axial orientation is determined by the concept of range equation, i.e. the first structure the U/S beam encounters will be the first structure to be displayed in the near zone, the last structure encountered by the U/S beam will be the last to be displayed in the far zone.

The lateral orientation will be determined by the direction of the dot (marker or knob) on the side of the probe, when the sonographer directs this dot or marker in the direction of a structure, this structure will be displayed on the same side of the dot shown on the screen. (See echocardiographic views)

Doppler U/S :

These are ordinary U/S waves, sent either in a pulsatile or a continuous manner, and are directed to the blood flow. The received signals are introduced to the Doppler shift equation (see before), and the resulting values are either illustrated in the form of velocities over a graph (see before), or are illustrated in terms of velocities in the form of color encoded dots (see before).

Tissue Doppler :

Tissue Doppler applies all the concepts of the classic Doppler but it is designated to be sensitive enough to elicit the velocities of the tissues. This is done by the use of "filters" set to detect the tissue's lower velocities and higher amplitudes.

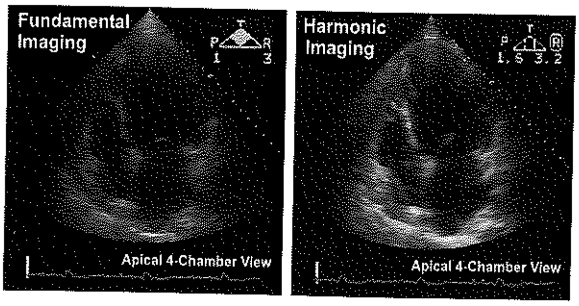
By applying the sample volume of the tissue Doppler over a selected tissue area, the probe will send U/S pulses of a known frequency to this area, then these pulses will reflect back with a difference frequency. So the machine can apply the Doppler shift equation and calculate the velocity of the reflector in the same way as before.

Strain, strain rate and speckle tracking are still not available for routine use in Egypt and many countries in the middle east , so these modalities are beyond the scope of this book.

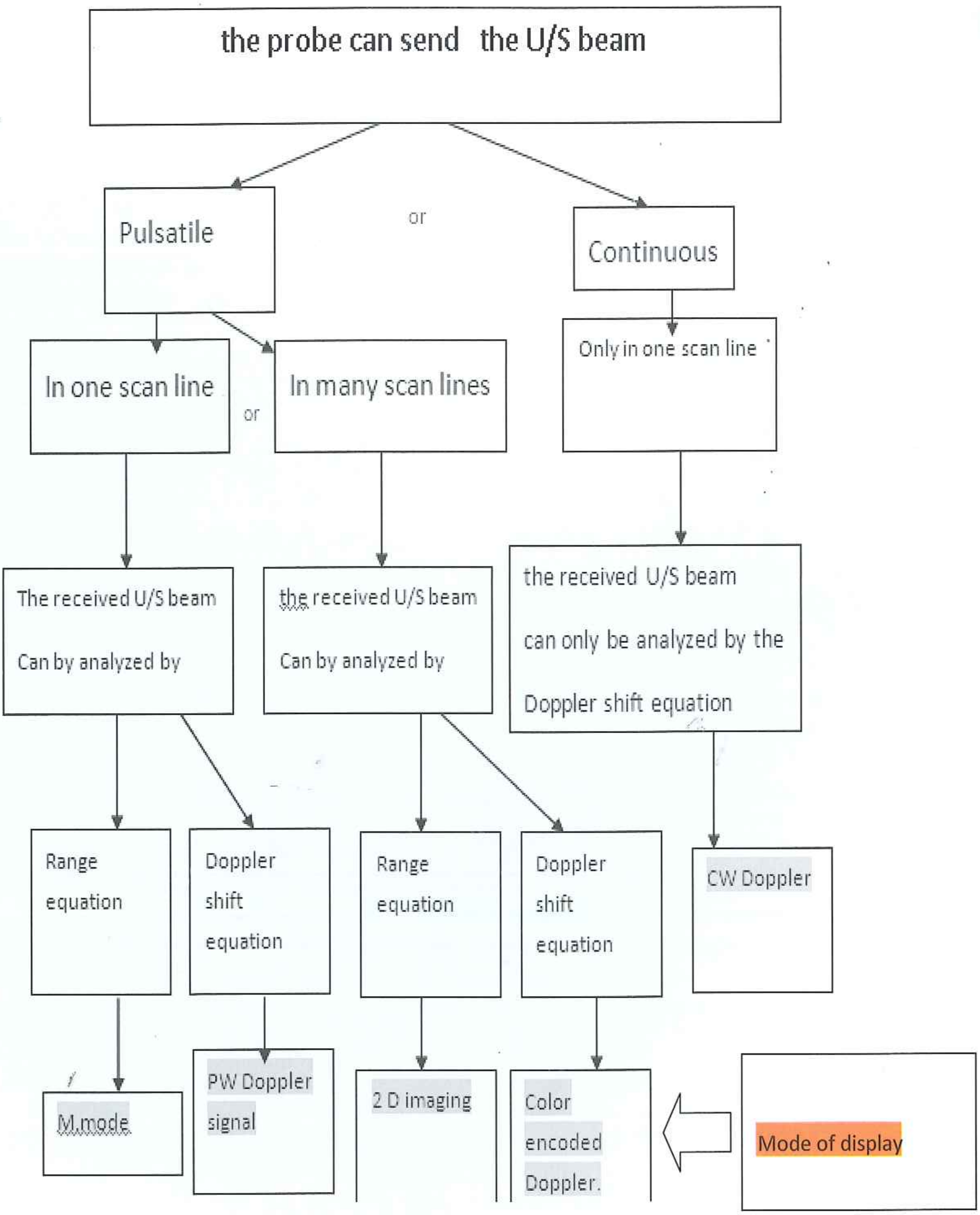
Contrast echocardiography agents are not available in Egypt and most of the middle east countries, so contrast echocardiography is beyond the scope of this book.

HARMONIC IMAGING

Modern ultrasound machines tend to use two different imaging modes: fundamental imaging and harmonic imaging. In fundamental imaging, the ultrasound transducer listens for the returning ultrasound at the same frequency at which it was emitted. However, ultrasound can cause tissues to vibrate at frequencies that are multiples of the frequency of the original ultrasound pulse. The transducer can thus be set to listen at a frequency that is higher (by a multiple) than the original frequency. Harmonic imaging allows for improved resolution of tissue interfaces, in particular, the endocardial border. Harmonic imaging is an option on many modern ultrasound machines. However, image quality is not always improved by harmonic imaging, and in some patients, fundamental imaging provides better overall image quality



The EMSE Physics back bone algorithm



Chapter 5 : Hemodynamics (Haemodynamics)

In this chapter we will discuss the concept of hemodynamics that will be applied to all valvular lesions and other main equations frequently used in echocardiography.

Remember :

- the normal LV systolic pressure is 120 mmHg
- Normal LA pressure is about 5 mmHg
- Mitral regurgitation is a systolic flow, so blood will regurgitate from the LV to the LA with high velocity due to the big pressure difference.
- The Normal aortic pressure during systole is the same as the LV during systole given that there is aortic stenosis. In cases of aortic stenosis the LV pressure will increase and the aortic pressure will decrease, this will lead to a high velocity jet. (the same concept applies to normal and stenotic mitral valve)
- In cases of normal mitral valve the LA pressure is nearly the same as LV pressure during the diastole.
- In cases of mitral stenosis the LA pressure will increase, this will lead to a high velocity jet into the LV.
- The normal aortic pressure during the cardiac diastole declines gradually from 120 mmHg to 80 mmHg.
- Normal LV end diastolic pressure is about zero.
- Aortic regurgitation is diastolic, so blood will regurgitate from the aorta to the LV with high velocity due to the big pressure difference.

Peak (maximal) Pressure gradient *(PPG) :*

As a general concept; what will happen if there is a stenosis across a tube containing a flowing non-compressible fluid ? Simply the pressure will increase before the stenosis and a high velocity jet will be generated after the stenosis, the popular scientist named professor **Daniel Bernoulli** had described this relationship on the basis of what is called energy conservation theory (different from the mass conservation theory) , that the fluid will keep its energy from being wasted when transferring across the stenosis and this energy will be transferred to a high velocity jet, so to clarify, the stenosis will generate a high pressure gradient that will lead to a high velocity jet. Bernoulli also stated an equation that converts the generated high velocity back to P.G, to clarify , for any high velocity jet we can measure the P.G. The equation he invented mainly was applied for engineering purposes and included many variables as friction factor and viscosity, however these variables are negligible to us when discussing the fluids within our biological body.

Remember that to detect velocities we use the Doppler U/S waves, to convert these velocities to P.G we use Bernoulli principal.

Simplified Bernoulli equation: $P.G = 4(V)^2$

Normally we have intra-cardiac valves that opens with full excursion (see diagram 33), so there will be minimal or negligible PG across any valve, however this negligible PG is mandatory for life in order for the flow to occur and for the valve to open.

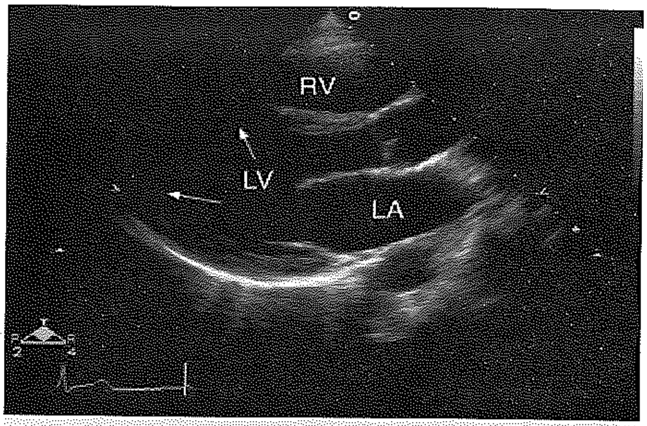


Diagram 33 : normal mitral valve showing full excursion of the mitral valve, creating low pressure gradients.

Normal trans-valvular flow :

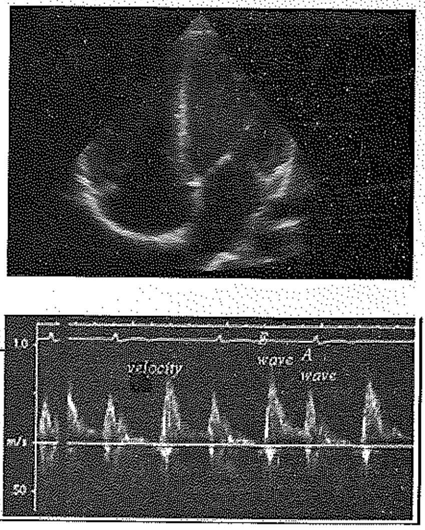
The general rules :

1) Normally no high velocity jets should be detected
2) Normal velocity across any valve should not exceed 1m/s (except in a hyperdynamic circulation)
3) Any high velocity jet is mostly abnormal.
4) Simplified Bernoulli overestimates the PG with velocities less than 1.7 m/s, and in the case of low velocity the echocardiographer can either describe the flow in terms of velocity or use the full Bernoulli equation {$\Delta P = 4\,(V_2)^2 - 4\,(V_1)^2$ }

Ex 1 : normal mitral flow :

Normally the mitral flow occurs on two phases, early filling phase " E wave " (passive filling phase) and then atrial contraction phase "A wave" , thus, to quantify the mitral velocities we must use the Doppler principle (see clinically applied Physics), where simply the Doppler equation describes flow in terms of velocities, remember that you must adjust the view in order to encounter the Doppler U/S wave parallel to the blood flow, typically this will occur in all apical views.(see diagram).

Diagram 34



Notice the typical normal velocity

Should be 1 m/s or less.

And remember that in cases of increased flow across the valve the velocity may increase without the presence of stenosis.

Doppler study aligned over the M.V

As the probe sends waves with known frequencies across the mitral valve and then these waves will be reflected back from the mitral flow but with different frequencies, by introducing all these information to the Doppler shift equation (built inside the machine) the machine will calculate velocities across the mitral valve. From an apical position, the blood is typically flowing towards the apex (towards the probe) so it will give a positive Doppler shift that will be displayed on the graph as positive deflection. See diagram 34.

Notice that typically normal velocities is about 1 m/s

N.B minimal or mild mitral regurgitation signal may be seen in healthy individuals. (see regurgitation jets)

The P.G for the E and A waves can be calculated using Bernoulli's equation (see diagram), where PG = $4V^2$, but it will be of little clinical value and instead we use the Mean Pressure Gradient. (See later)

Ex 2 : Normal tricuspid flow:

Using the same concept, you can quantify the tricuspid flow by aligning Doppler flow towards the tricuspid valve in an apical 4 chamber view, PLAX in flow view and PSAX great vessels level.

The normal tricuspid flow is the same as the mitral flow.

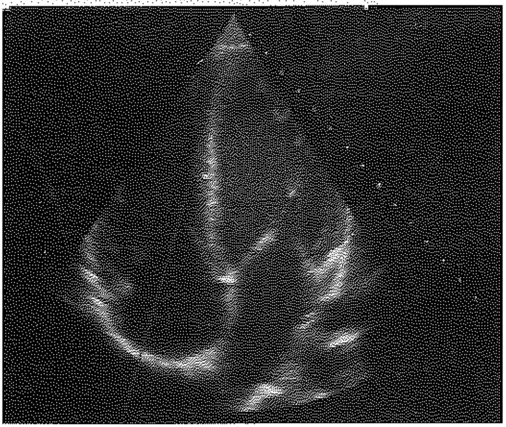
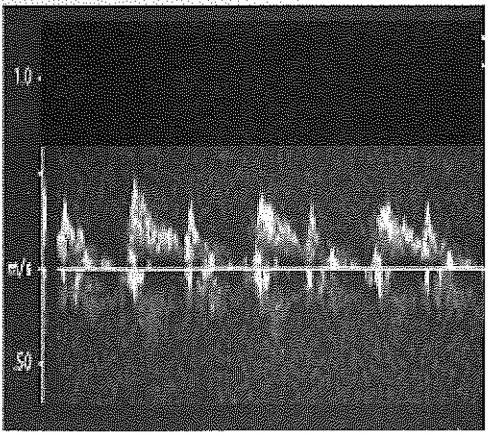


Diagram 35



Notice the typical normal velocity

Should be 1 m/s or less.
However normally the TV velocities are lower than M.V velocities.

Ex 3 : Normal aortic flow

By aligning the Doppler flow across the aortic valve in an apical 5 chamber view, the Doppler shifts occurring will be negative Doppler shifts giving negative wave signals because the blood is flowing away from the apex towards the LVOT, aortic valve and the ascending aorta. As we have only one systolic contraction so we will only see one negative deflection.

See diagram 36, notice again that the velocity is about 1 m/s

As soon as the velocity is calculated then the PG can be calculated. PG = $4V^2$ (this is done automatically by the echocardiography machine)

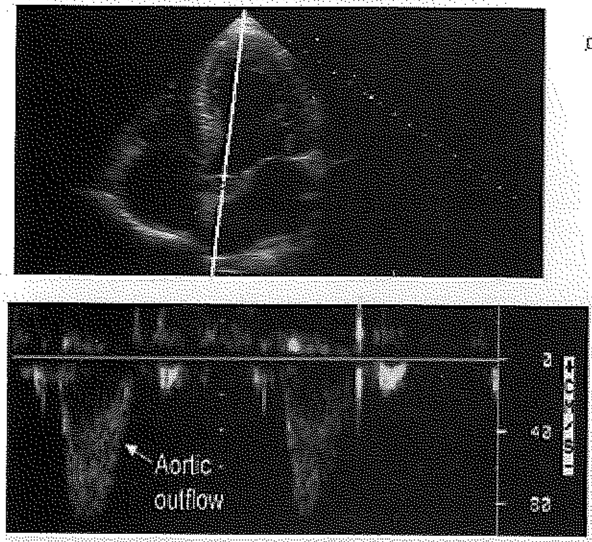


Diagram 36

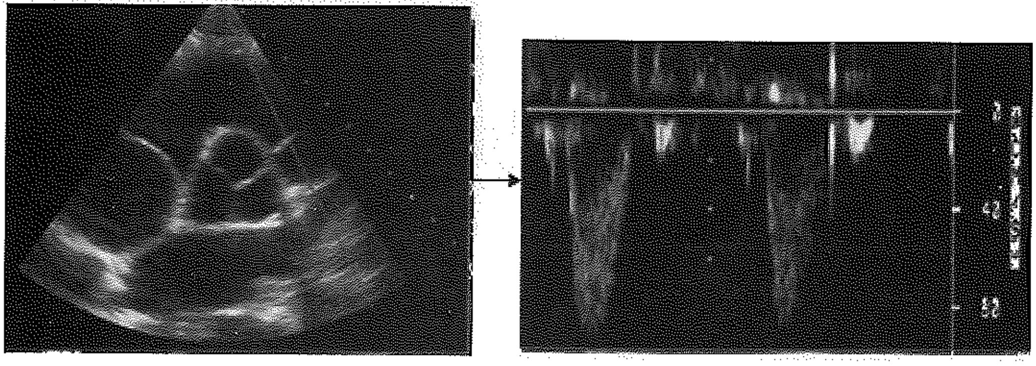
Notice the normal low velocity jet aortic flow. Reaching < 100 cm/s

---

Ex 4: Normal pulmonary flow

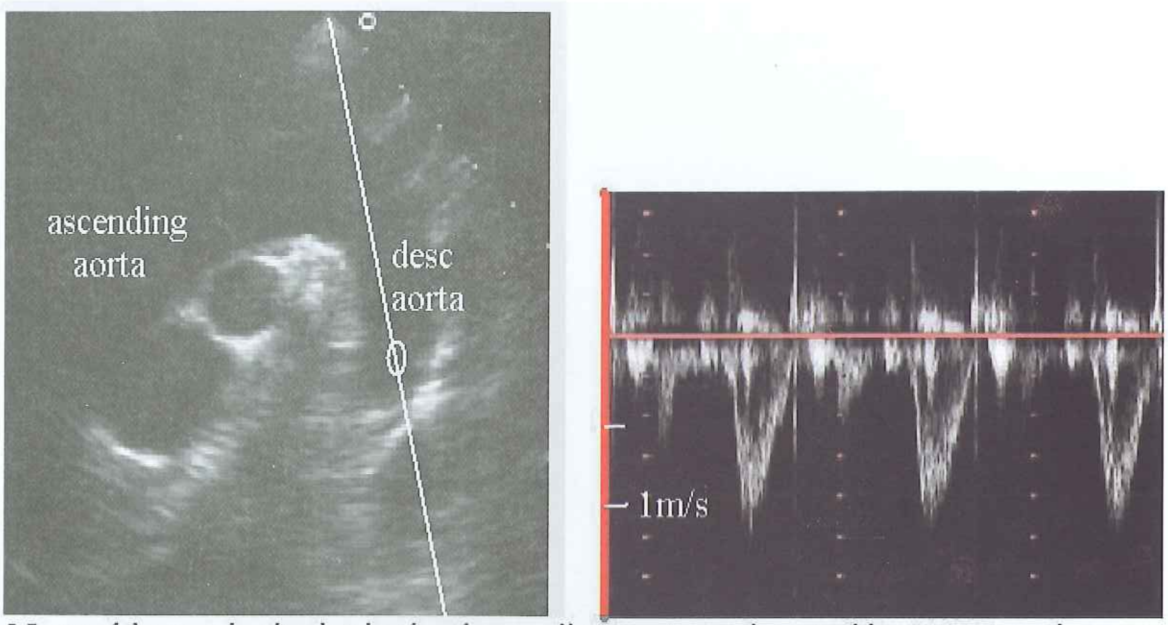
By aligning Doppler wave over the pulmonary valve in either PLAX outflow view or PSAX great vessels level, pulmonary flow will appear the same as aortic flow. (the blood will normally flow from the RV to the pulmonary artery (PA), i.e. away from the probe, so normally the PA flow will give a negative deflection.)

Diagram 37 A



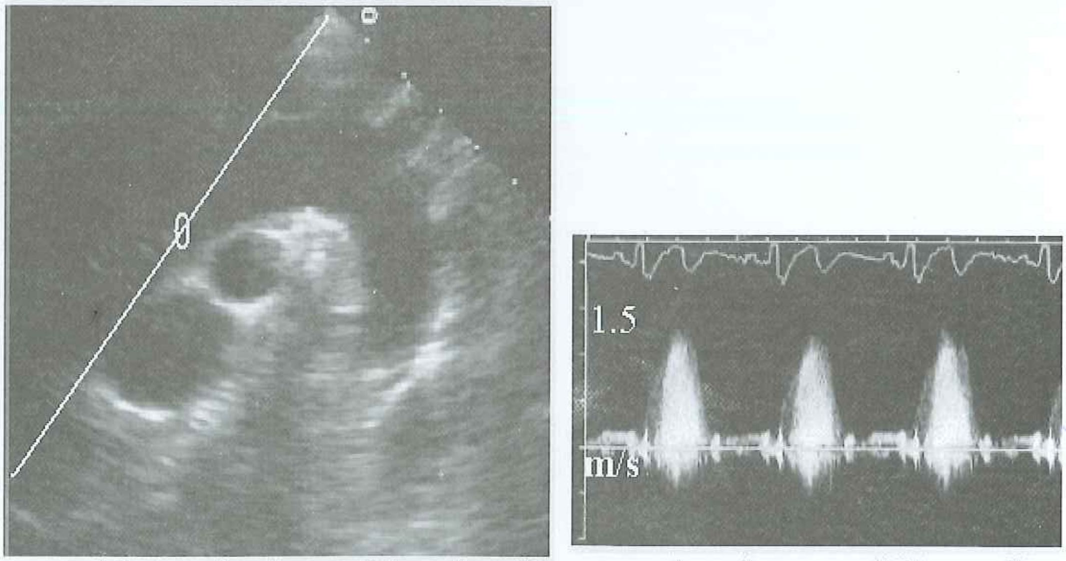
Normal flow in the ascending aorta and descending aorta:

Diagram 37 B



Normal low velocity jet in the descending aorta as detected by CW Doppler. normally the blood flows away from the probe leading to negative Doppler shift.

Diagram 37 C



normal low velocity positive signal representing the normal forward flow in the ascending aorta.

Examples for an abnormal high velocity jet :

Notice the high velocity mitral flow, reaching 2.6 m/s

PPG = 4 v^2 → PPG= 4 $(2.62)^2$

→ PPG = 27.5 mmHg

(the equation is automatically calculated by the machine)

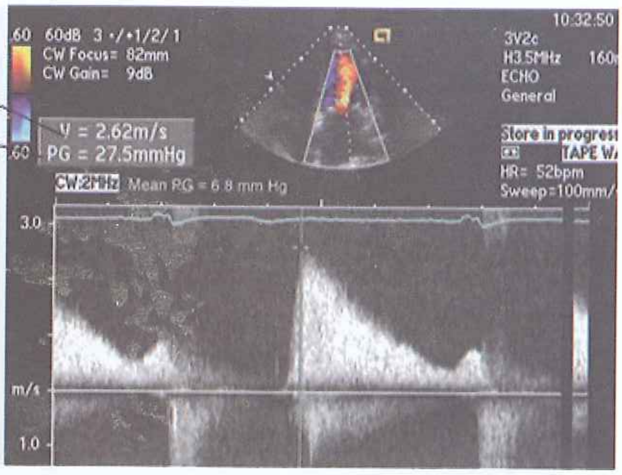
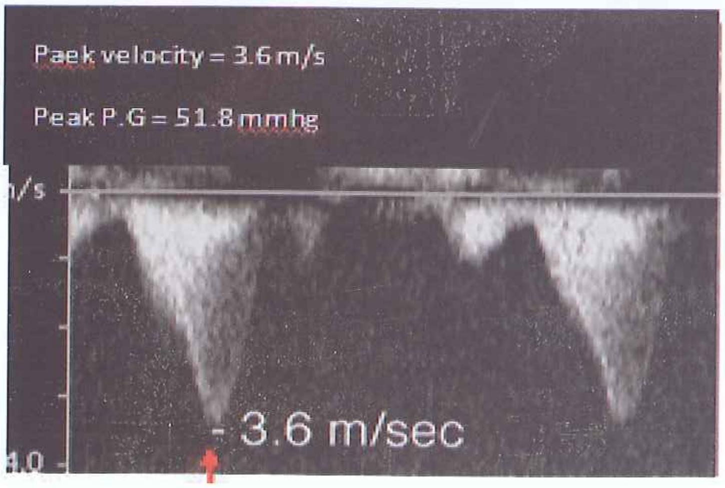


Diagram 37 B Trans mitral Doppler image recorded in a patient with concurrent mitral stenosis and mitral regurgitation. Note the high peak early gradient (27.5 mm Hg) but the rapid decay and a negligible pressure gradient at end-diastole. Compare the peak early gradient of 27.5 mm Hg with the mean gradient of only 6.8 mm Hg. This discrepancy between peak and mean pressure gradient is often seen in patients with concurrent mitral regurgitation.

Diagram 38 : Increased trans aortic velocity



The same concept of Bernoulli's equation is also applied here , notice that the PPG = $4v^2$

51.8 = $4(3.6)^2$

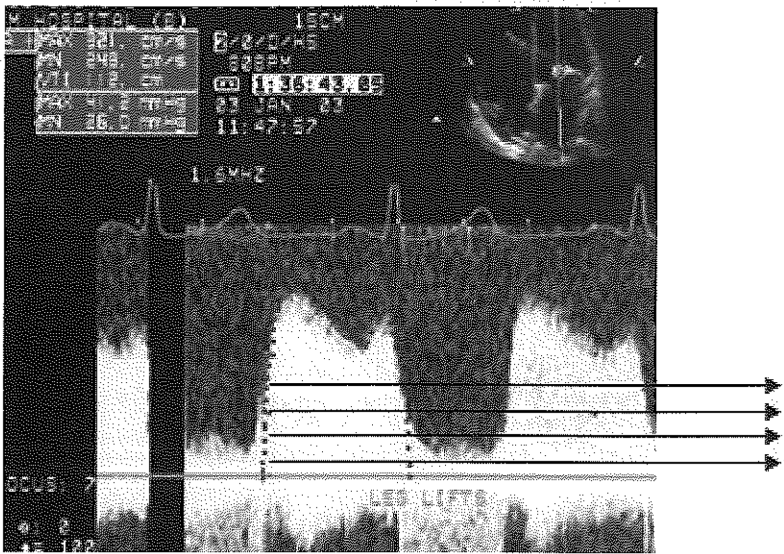
Mean pressure gradient:

Instead of measuring the Peak P.G (i.e. only one point where the flow peaks) a more accurate measure can be calculated, namely the Mean P.G.

This is done by tracing the signal acquired from the desired valve where the machine converts every point of the signal into a PPG then by adding all these PPG together then dividing them by their number, a mean value is obtained. See diagram 39.

Diagram 39: mean PPG for mitral valve flow.(normally the MPG should be less than 2 mmHg)

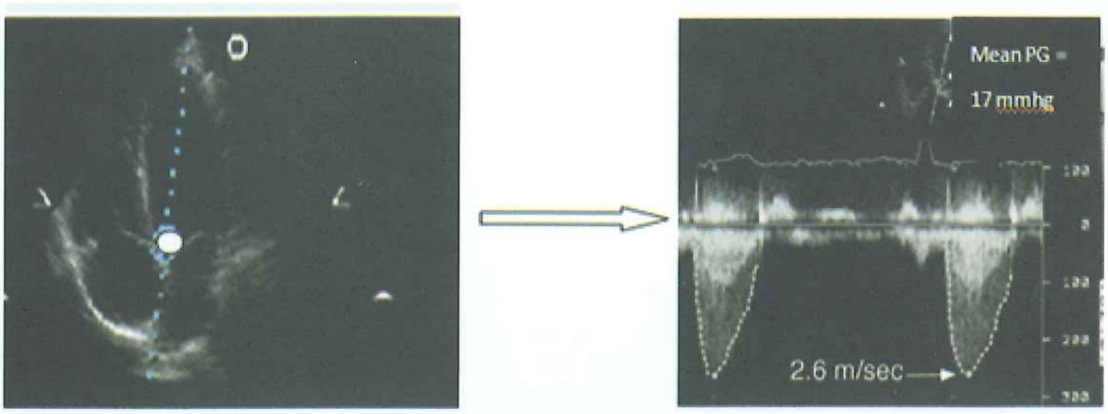
Diagram 39: mean PPG for mitral valve flow.(normally the MPG should be less than 2 mmHg)



Each dot in the flow is calculated as a PPG then summated then divided by their number; this is done automatically by the machine when the echocardiographer traces the signal.

Mean PG is better validated during the assessment of a case of hyper dynamic circulation, as we measure the mean change of the PG rather than only one peak value.

Diagram 40: MPG for aortic flow. (Normally the MPG for the aortic valve should not exceed 8 mmhg)



N.B : it is very important to remember that all measures should be averaged over 5 beats (10 beats in A.F) and any ectopic or post ectopic beats should be ignored

N.B the mean PG can be estimated by the following equation : Mean PG = (PPG/1.45) +2

N.B : the simplified Bernoulli's equation tends to overestimate the PPG when applied to a non stenotic valve when compared to the direct pressure measurements using intra cardiac catheterization.

When examining a normal valvular flow it is better to report in terms of velocity.

A professional echocardiographer only translates the velocity to PPG in cases of increased velocities.

Velocity time integral (VTI) :

The rational for the calculation of the velocity time integral is that the peak Velocity (not the peak P.G) did not give accurate information about the flow, so it must be integrated to time.

For example, if two types of flow have the same peak velocity (ex 4m/s) but flow "A" takes more time than flow "B" (see diagram 41), will the peak velocity describe an accurate relationship when comparing both types of flow? No it will not, rather, integrating the time as a variable will accurately describe the flow. To do this the Area under the Curve"AUC" must be calculated. The machine divides the signal flow to many small rectangles, then by calculating the area of each rectangle then integrating them together (i.e. summating them) , the AUC will be calculated.(remember that area of rectangle = length X width)

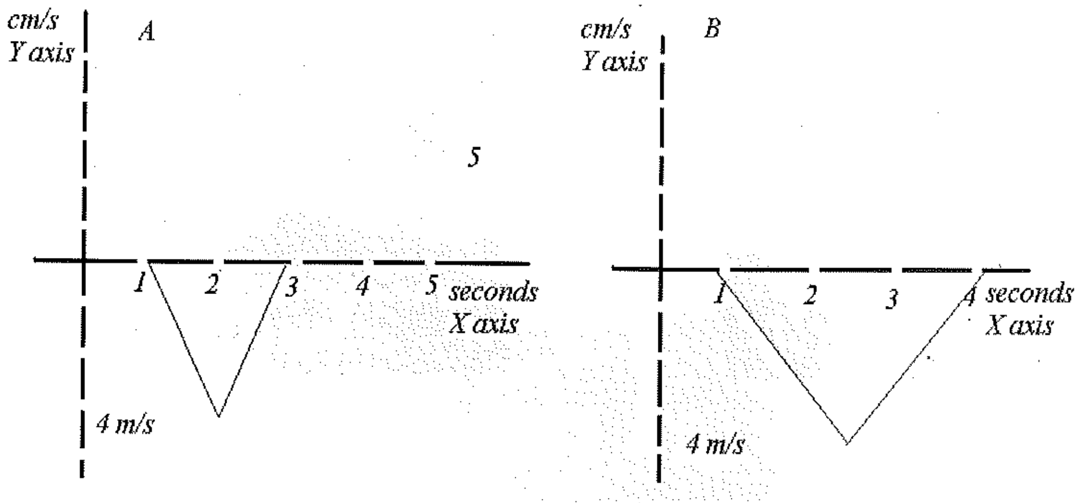
But it is very crucial to understand the definition of the VTI. By multiplying the X axis by the Y axis, the resulting number will be in centimeters, the X axis (width) is defined in seconds and the Y axis (length) is defined in cm/s, so VTI = L (cm/s) * W (s) = A (cm).

VTI : velocity time integral , L : length, W : width , A : area under curve.

So the AUC is not defined as cm^2 , and is not defined in cm/s, VTI is defined in cm.

See example in diagram 41.

diagramatic representation for trans aortic flow in two different cases



the two flows will be the same if compared by velocties, BUT flow "B" is more, if the time factor is considerd into the measurments, i.e. time is integrated into the velocity, this is done by measuring the VTI. see text for details.

diagram 41

Blood volume measurements using Doppler principle and mass conservation theory:

Now, what will happen if we multiply CM by CM$^{2?}$, the result will be in CM^3! , remember that this is a mathematical fact, so actually when we multiply the VTI of a certain flow by the cross sectional area in which it had flowed , the resultant value will by the volume of that flow. See diagram 42.

So the stroke volume of the left ventricle = VTI of the LVOT X cross sectional area of the LVOT.

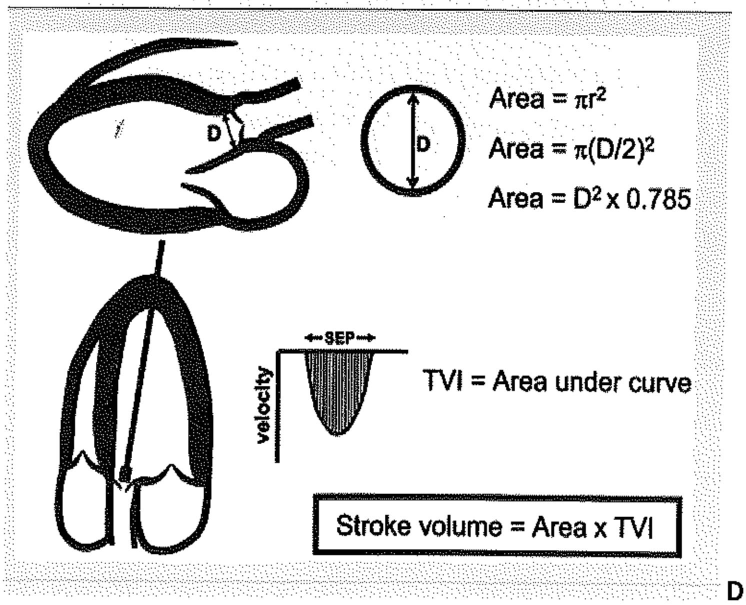


Diagram 42

To calculate the VTI of the LVOT we will use PW Doppler aligned inside the LVOT, and then by tracing the signal flow, the machine will not only measure the MPG but also will it automatically calculate the VTI.

To calculate the cross sectional area of the LVOT, measure the diameter of the LVOT, as the LVOT is cylindrical in shape so its cross sectional geometry will be a circle, the area of the circle = πr^2 , this is the diameter of the LVOT divided by two then squared then multiplied by 3.14.

See illustrating diagrams 42 and 43.

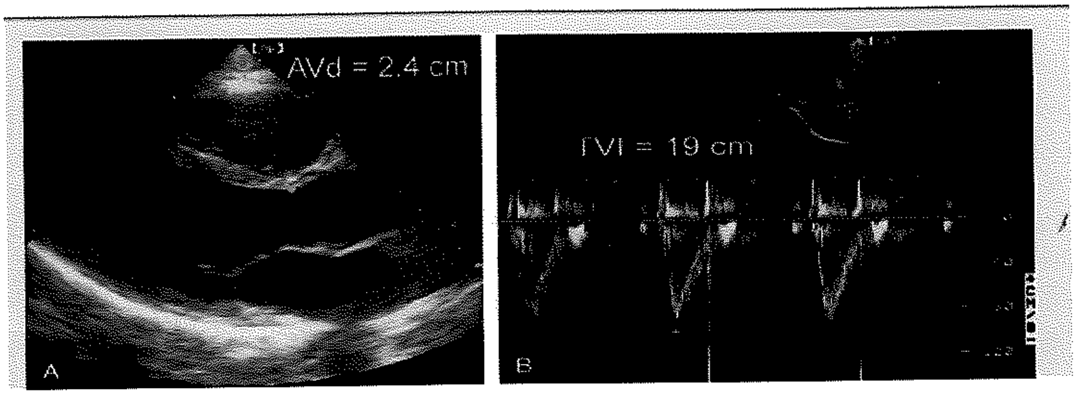


Diagram 43, SV $=\{ (2.4/2)^2 \times 3.14 \} \times 19$

SV = 85.9 cm^3

According to the concept of mass conservation theory, the volume of blood traversing the LVOT equals the volume of blood traversing the aortic valve (notice that this will be over the expense of the increased velocity of the blood through the aortic valve to keep its volume unchanged).

This concept will be used to calculate the aortic valve area by the continuity equation (see later)

Volume of blood traversing the LVOT= volume of blood traversing the aortic valve.

VTI LVOT (PW) X area of LVOT = VTI aortic valve (CW) X area of the aortic valve.

Stroke volume = VTI aortic valve (CW) X area of the aortic valve.

Aortic valve area = stroke volume / VTI aortic valve.

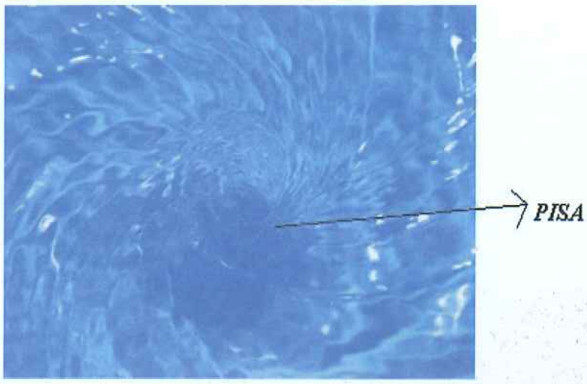
By the same concept the volume of blood filling the left ventricle will be calculated by multiplying the VTI of the mitral flow by the cross sectional area of the mitral valve.

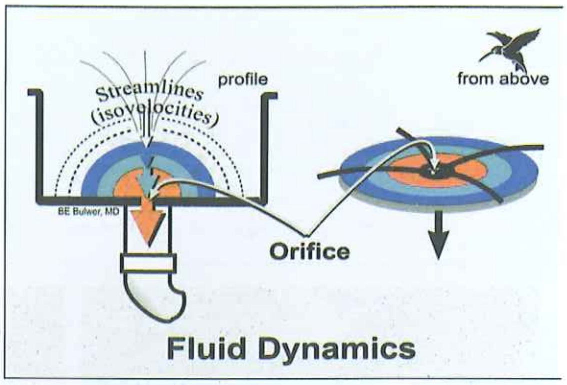
Regurgitation volume, regurgitation fraction, proximal iso-velocity surface area and effective regurgitation orifice area are not commonly used in clinical practice so they are briefly discussed in this book.

The concept of the proximal isovelocity surface area "PISA" convergence zone:

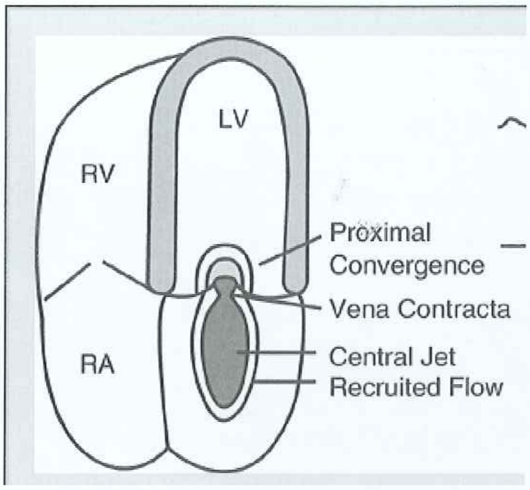
Imagine a container filled with a non compressible fluid. If we open a hole down in the container, the fluid will have 3 stages while flowing out; the swirling zone just before the hole, the part of the flow exactly through the hole, and finally the part of the flow after leaving the hole.

if the hole is so big, what will happen to the swirling zone just before the hole ?, it will increase in diameter, and vice versa.





Let's give an example in real echocardiography, in a case of mitral regurgitation, the blood flow will regurgitate from the LV to the LA through the mitral valve. This flow will have 3 stages while flowing out of the LV and into the LA; the swirling zone just before the valve, the part of the flow exactly through the valve, and finally the part of the flow after leaving the valve (i.e. the part of the blood flow actually in the LA).



Schematic demonstration of the principal of PISA. The various components of the true regurgitant signal are outlined on the schematic, including the proximal flow convergence zone, the vena contracta, and a central high-velocity jet surrounded by a lower velocity recruited flow.

N.B: All the components of the flow can be used to assess the severity of the regurgitation jet.

Severe regurgitation means that the flow regurgitates from a wider orifice (e.g. poor cooptation of the MV), this will lead to a larger PISA & wider vena contracta.

N.B: The PISA flow has relatively much lower velocity than the actual regurgitation jet inside the LA.

To elicit the PISA flow the echocardiographer has to set the Nyquist limit to a lower value (40 cm/s).

If the regurgitation zone is circular, the PISA will be hemispherical in shape.

The area of the hemisphere is $2 \pi r^2$.

What will be the resultant value of the multiplication of the area of the proximal convergence zone (PISA) "defined in cm^2" **by** the Nyqiust velocity "defined in cm/s" at which the PISA flow was elicited? The resultant number will be $cm^2 \times cm/s = cm^3/s$, which is defined as the flow rate.

Flow rate = $2 \pi r^2$ X aliasing velocity

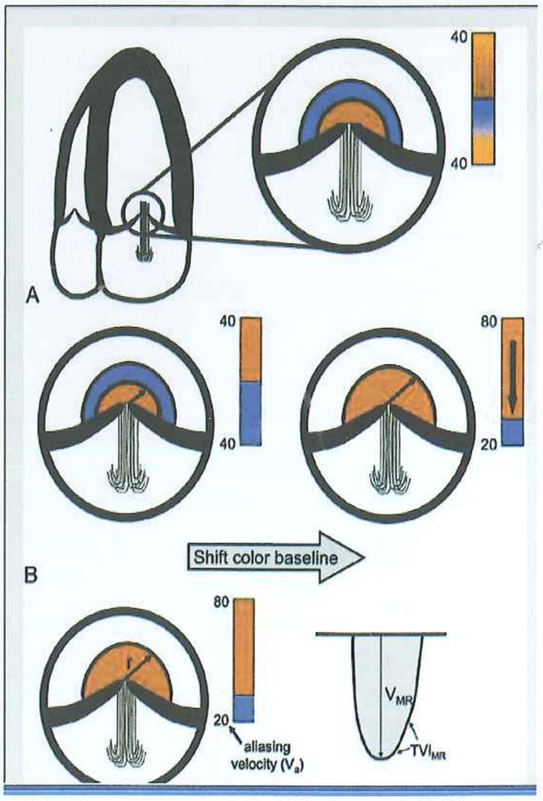
According to the mass conservation theory, the PISA flow rate must be equal to the regurgitation flow rate through the mitral valve.

PISA Flow rate = effective regurgitation orifice area "EROA" (Cm^2) X peak velocity of the mitral regurgitation (cm/s)

Therefore : EROA = PISA flow rate / peak MR velocity

Regurgitation volume = EROA (cm^2) X VTI of the MR (cm)

Regurgitation volume will be defined in cm^3



$$Flow = 2\pi \times r^2 \times V_a$$

$$ERO = Flow \div V_{MR}$$

$$RV = ERO \times TVI_{MR}$$

Pressure half time:

The pressure halftime is defined as the time needed to the pressure to fall to its half value. For example, if the LA pressure is 5mmHg so when the mitral valve opens the pressure will be divided on two (as we have two chambers, and given that the LV end diastolic pressure is zero).

So if the mitral valve area is reduced i.e.: a stenotic lesion, the time taken for the pressure to fall to its half value will be prolonged. (this is the same concept if we have two balloons connected to each other from the neck, one is inflated and the other is not, and the neck is closed, if we opened the neck widely the pressure inside the inflated balloon will drop to its half value. i.e. the pressure will be divided by two, and this will occur in a relatively short time. BUT , if we do not open the neck inbetween them widely, i.e. small neck area, the pressure will be divided by two as before but in a relatively longer time.)
The equation to describe this relationship between the PHT and the valve area is MVA= 220/PHT. So, PHT prolongation indicates a smaller MV area.

Another situation where the PHT seems rational to use is in the assessment of the aortic regurgitation, in aortic regurgitation the blood flows back to the LV, so typically, when the case is mild, the blood regurgitates from a relatively small orifice, so the time to the pressure to fall to its half value will be relatively longer than in severe cases where the blood regurgitates back from a larger area with relatively shorter PHT.
To summarize, when the flow traverses through a relatively wide area it will pass with short PHT and vice versa.

The three EMSE rules that must be assumed for accurate usage of pressure half time calculations:

1)Chronic pressure changes, 2)Normal LV diastolic pressure . 3)Normal LV diastolic compliance.

1) The echocardiographer should never use the Pressure half time method to asses any lesion in cases of acute or rapid pressure changes:

- Do not use the pressure half time to estimate the mitral valve area post valvotomy.
- Do not use the pressure half time to estimate aortic regurgitation severity in acute cases.

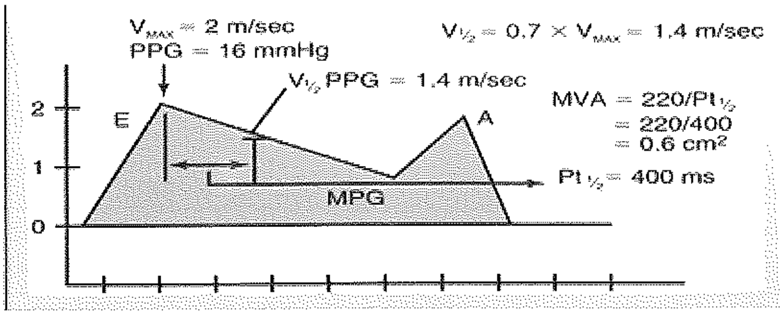
2)The pressure half time derived calculations depends on the fact that *the left ventricular diastolic pressure is zero* . So any factor that elevates the Left ventricular diastolic pressure will shorten the pressure half time.

- Do not use the pressure half time calculations to assess the mitral valve area in cases of aortic regurgitation or grade III diastolic dysfunction (high LV end diastolic pressure).
- Do not use the pressure half time to estimate aortic regurgitation severity in very late cases (due to the concomitant high LV end diastolic pressure).

3)The pressure half time derived calculations depends on the fact that the left ventricular diastolic *compliance* is normal.

- So, do not use the pressure half time to calculate the mitral valve area in cases of grade I diastolic dysfunction, otherwise this will yield falsely prolonged PHT.

Diagram 44 A



PHT : is the time needed for the pressure to fall to it's half value.

So we have to calculate the time interval for the pressure to fall from 16 mmHg { $(4(2)^2$ } to 8 mmHg { $4(1.4)^2$ }

So we have to calculate the time for the pressure to fall from 2 m/s to 1.4 m/s. this time interval is 400 msec

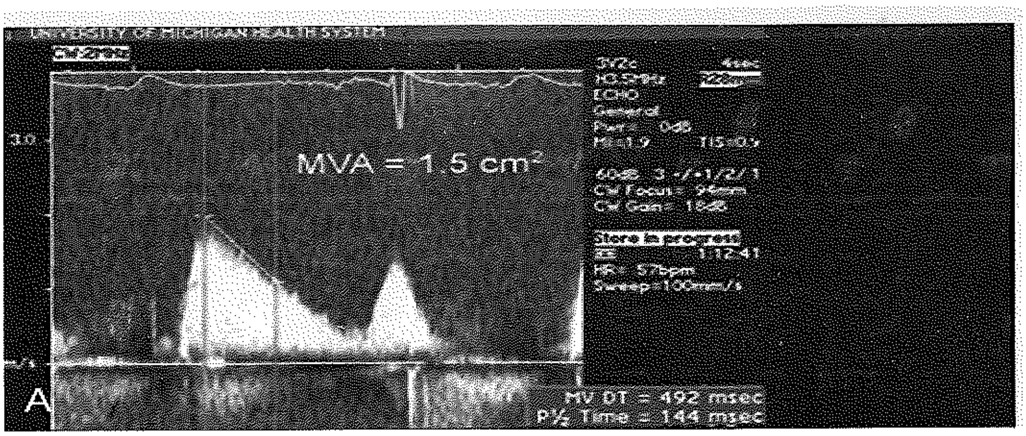


Diagram 44 B, notice the slope drawn by the echocardiographer.

The machine will automatically calculate the MVA by : MVA= 220/ PHT

Chapter 6 : Valvular stenotic lesions

To quantify any stenotic lesion : you must quantify the valve area and the pressure gradient across this valve.

To asses any lesion : you must use 2 D echo, M.mode and Doppler.

N.B : To quantify any regurgitation lesion : you must quantify by color Doppler how much the regurgitation jet occupied from the chamber it regurgitated to.

Mitral valve stenosis :

Normal Mitral valve: Normally the mitral valve apparatus is formed of annulus, leaflets, chordae, papillary muscles and the L.V walls to which the papillary muscles are attached to.

All these structures normally appear thin with normal echogenicity with no calcifications or fusion.

The normal mitral valve area is 3-6cm^2.

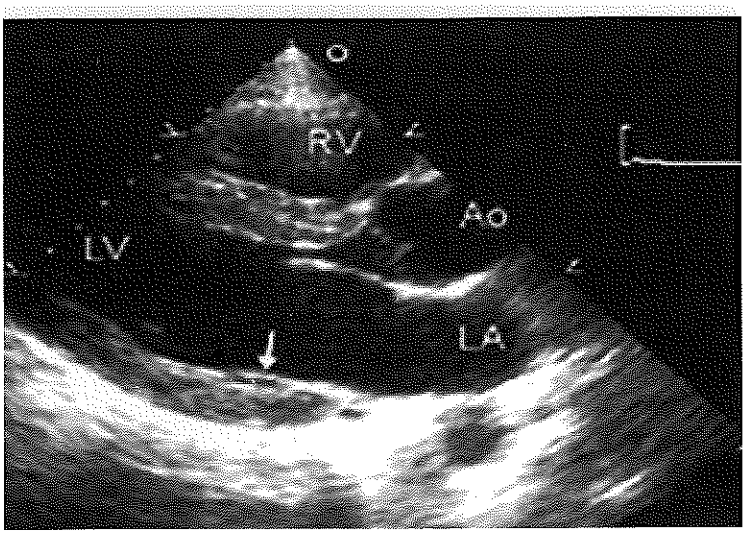


Diagram 45 showing normal mitral valve in end diastole.

The posterior mitral leaflet makes a crescent over the anterior mitral valve leaflet, but the distance from the annulus to the closure line is longer in the anterior leaflet than in the posterior leaflet. The end result is that both leaflets have the same surface area. See diagram 46.

The mitral valve is formed of scallops "pieces or parts". See diagram 46

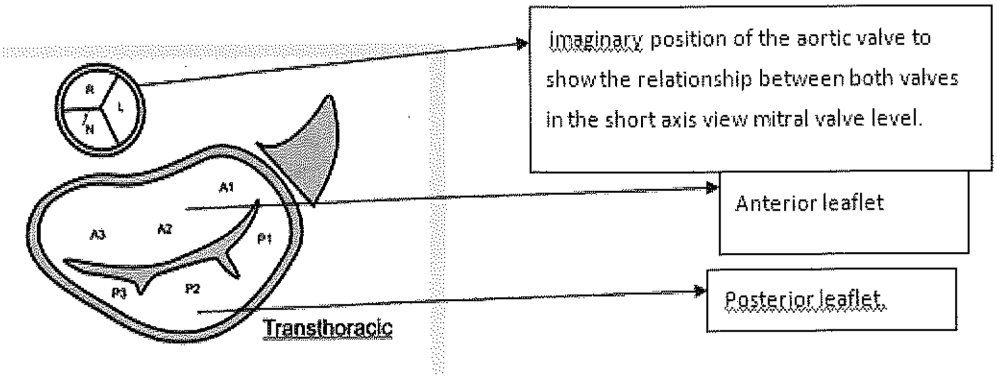

imaginary position of the aortic valve to show the relationship between both valves in the short axis view mitral valve level.
Anterior leaflet
Posterior leaflet
R
L
N
A1
A2
A3
P1
P2
P3
Transthoracic

Diagram 46 : showing different parts (scallops) of the mitral valve.

Mitral stenosis "MS":

Causes of MS :

Causes of Mitral Stenosis

- Rheumatic: most common cause
- Congenital
  - Parachute mitral valve: single papillary muscle to which chordae to both leaflets attach; results in mitral stenosis or mitral regurgitation
  - Supravalvular mitral ring
- Systemic diseases: can cause valvular fibrosis
  - Carcinoid
  - Systemic lupus erythematosus
  - Rheumatoid arthritis
  - Mucopolysaccharidosis
  - Healed endocarditis
  - Prior anorectic drug use
  - Severe mitral annular calcification

Echocardiography findings :

N.B : it is very important to remember that all measures should be averaged over 5 beats (10 beats in A.F) and any ectopic or post ectopic beats should be ignored

2D echo: thickened leaflets with hyperechogenicity, may be areas of calcification.

Restricted motion, typically hook stick, appears in rheumatic pathology, and the commissures may be fused. see Diagram 47.

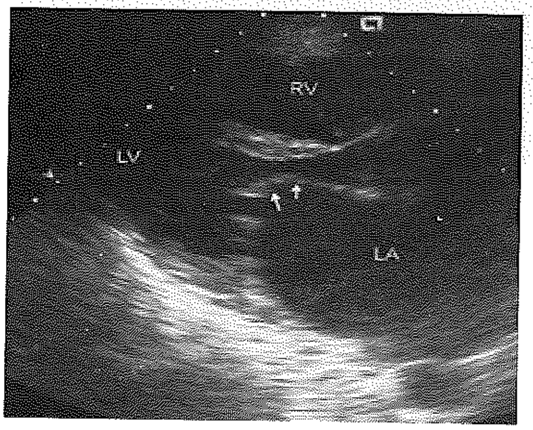
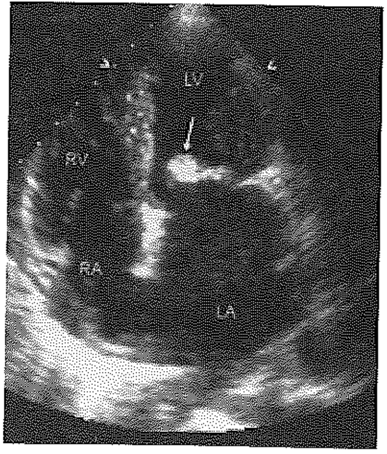


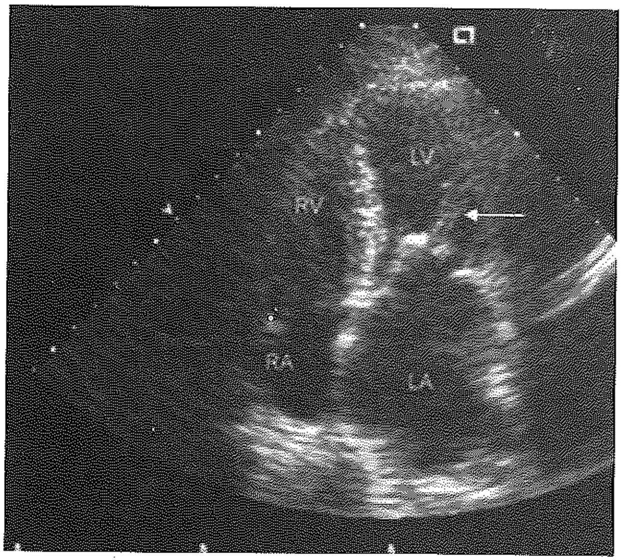
Diagram 47 (A)

Hook stick appearance shown by the arrows, also notice the thickened leaflets. (Compare to a normal M.V)



47 B Notice the spot of the calcification.

Sub-valvular affection may be associated in terms of thickening, fusion and calcification. These findings can be seen in any view showing the mitral valve.



47 (C) Notice the subvalvular thickening marked by the arrow.

Planimetry : direct measurement of the mitral valve area .

1) Bring a typical PSAX view mitral valve level.

2) Press the trace bottom.

3) Use the track ball to trace the mitral valve area as shown in diagram 48.

Planimetered area less than 2.1cm^2 indicates mitral stenosis (quantification is in the table below)

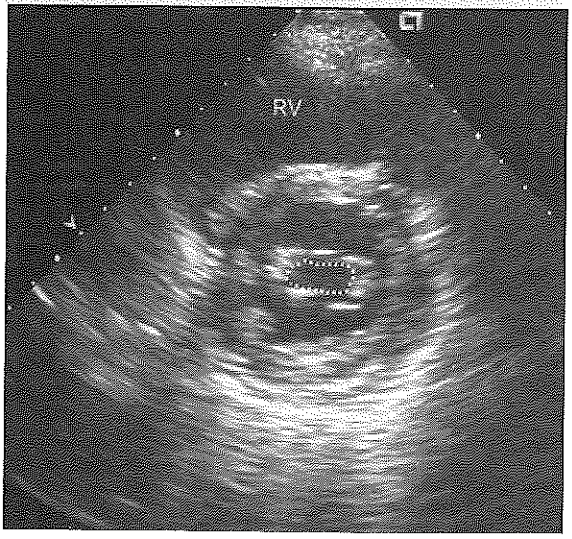


Diagram 48: Mitral valve area tracing in PSAX view.

Grading of severity of mitral valve stenosis by area in cm^2 by either planimetry or PHT :

Normal MV area	Anatomical stenosis without physiological effect	Mild mitral stenosis	Moderate mitral stenosis	Severe mitral stenosis
3-6 cm^2	2.1 - 3 cm^2	1.5 -2.1 cm^2	1-1.5 **cm^2**	Less than 1 cm^2

M.mode: thickened leaflets, flat E.F slope. This is the least valid mode to assess mitral stenosis. See diagram 49.

Diagram 49 A: normal mitral valve.

49 B: mitral stenosis. Notice the flat E-F slope and the thick leaflets (denoted by arrow) in a case of M.S

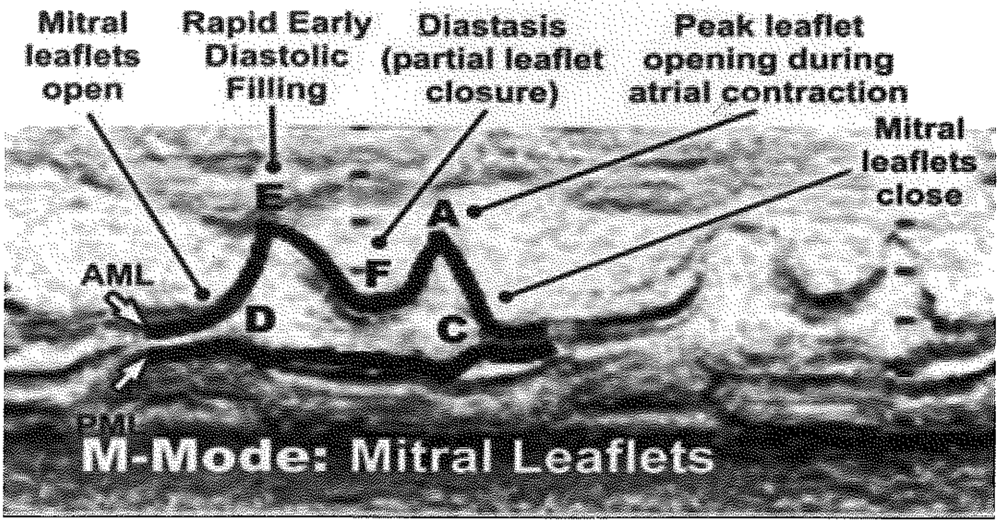

Mitral leaflets open
Rapid Early Diastolic Filling
Diastasis (partial leaflet closure)
Peak leaflet opening during atrial contraction
Mitral leaflets close
E
F
A
AML
D
C
PML
M-Mode: Mitral Leaflets

Diagram 49 A: Normal M.mode trace of the mitral valve.

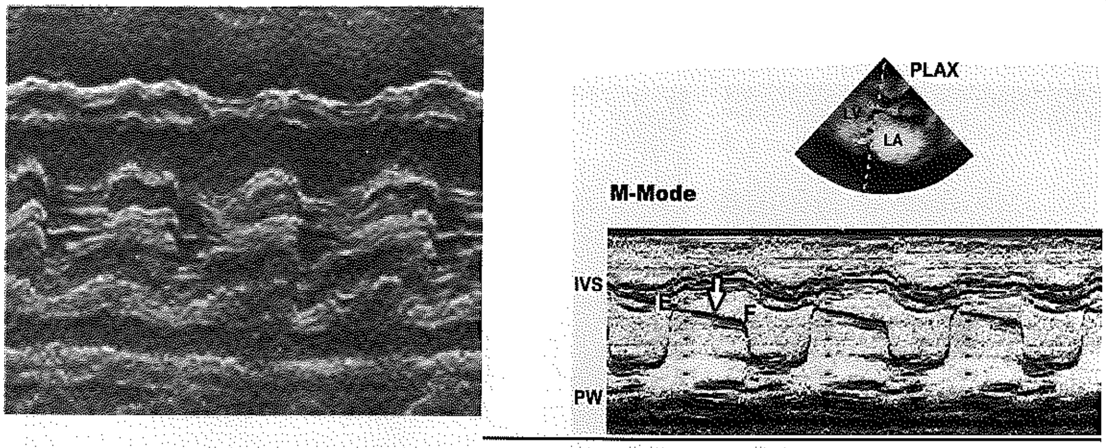


Diagram 49 B: mitral stenosis (M.S). notice the flat E-F slope and the thickened leaflets.

Doppler study: we will use the MPG, PHT and EPASP

1. Optimize a typical apical view, either an apical 4 or 2 chamber view.
2. Align the curser line through the mitral valve (using the track ball).
3. Put Doppler study (by pressing the PW or the CW Doppler button)
4. Freeze the screen.
5. Press the trace button & use the track ball to trace the signal as shown below.

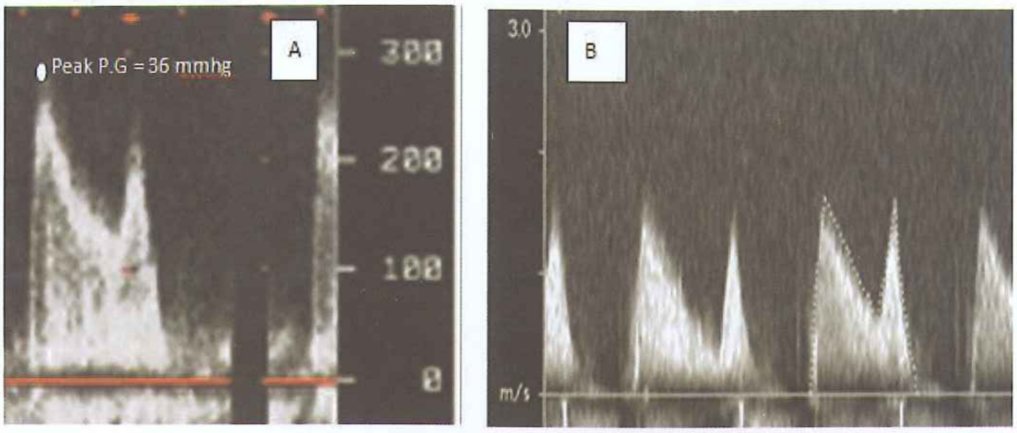
Both PW and CW can be used, in general, one school is to start with CW Doppler, if any high velocity jet is encountered, PW is used to localize its origin, either valvular or supra valvular.

A stenotic mitral valve will lead to a high velocity jet beginning at the valve and flowing to the supravalvular region. This high velocity jet can be detected by a Doppler study showing the conventional mitral in flow signal (see diagram), you can measure the Peak Pressure Gradient but it will be of less value in quantification of mitral stenosis. The mean pressure gradient (MPG) is a better measure (remember that the MPG is done by tracing the mitral inflow signal detected by the Doppler study). See diagrams 50 A, B& C.

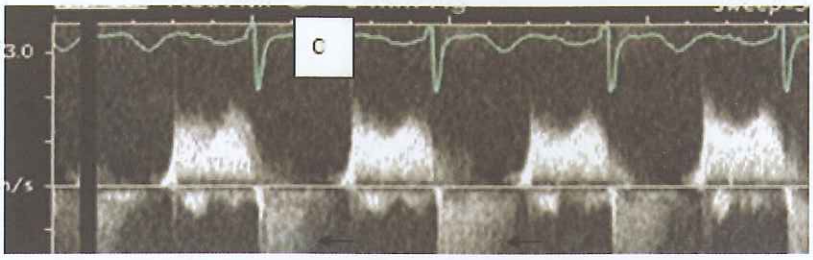
Remember that $PG = 4 * V^2$. A high velocity jet will lead to a high calculated PPG and MPG.

Diagram 50: Calculating the peak and mean PG's.

Diagram 50 : calculating the peak and mean PG



Examples for Peak pressure gradient and Mean pressure gradients (P.G) in two different patients.



Combined case of M.S and M.R. (aliased mitral regurgitation signal is denoted by the arrow, see later)

Mean P.G: Table of quantification of mitral stenosis (M.S) by M.P.G provided there is no hyperdynamic circulation.

Normal	mild M.S	Moderate M.S	Severe M.S
< 2 mmHg	2-5 mmHg	5-10 mmHg	>10 mmHg

To differentiate hyperdynamic circulation from M.S, the MV apparatus will be normal in 2 D echo in case of hyperdynamic circulation but abnormal in true M.S.

Hyperdynamic circulation will overestimate the MPG. So M.V area is better used.

Pressure half time is still validated in mild & moderate cases of hyper dynamic circulation.

N.B : in the presence of any motion restriction of the mitral valve plus a velocity of more than 1 m/s , mitral stenosis is diagnosed even if the values of the PPG and MPG lie within normal ranges.

- **Assessment of mitral stenosis from the estimated pulmonary artery systolic pressure EPASP :**

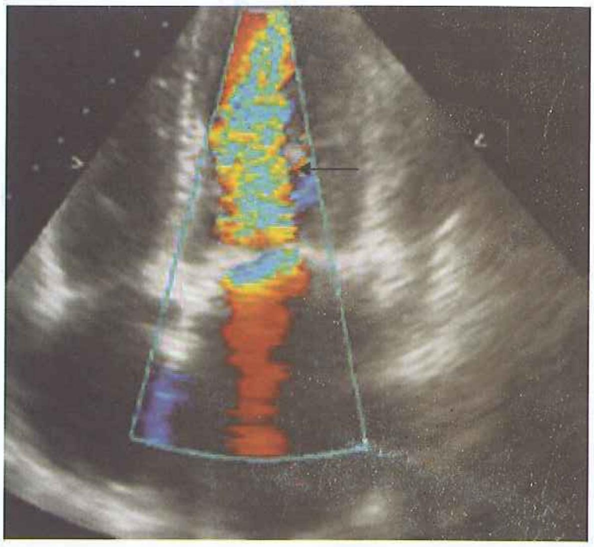
EPASP <30 mmHg → mild mitral stenosis.

EPASP 30- 50 mmHg → moderate mitral stenosis.

EPASP >50 mmHg → severe mitral stenosis.

Color Doppler:

Color Doppler will show a high velocity jet flowing into the L.V cavity, it is less valuable in quantification of mitral stenosis, but, it is important to track this high velocity jet of the mitral valve and to see if it is encountering the LVOT or not. See below.



Notice the high velocity mosaic pattern filling the LV cavity (denoted by the arrow)

Pressure half time "PHT":

Recall that the pressure half time is the time needed for the pressure to fall to its half value (for further illustration see hemodynamics).

Mitral stenosis will lead to prolongation of the PHT.

1) Perform the same steps of the Doppler study.
2) Press the pressure half time button.
3) Optimize the starting point over the peak E wave.
4) Align the enabled line over the deceleration slope of the E wave as illustrated in the diagram 51.
5) MVA (in cm^2) = 220/PHT (automatically calculated by the machine)

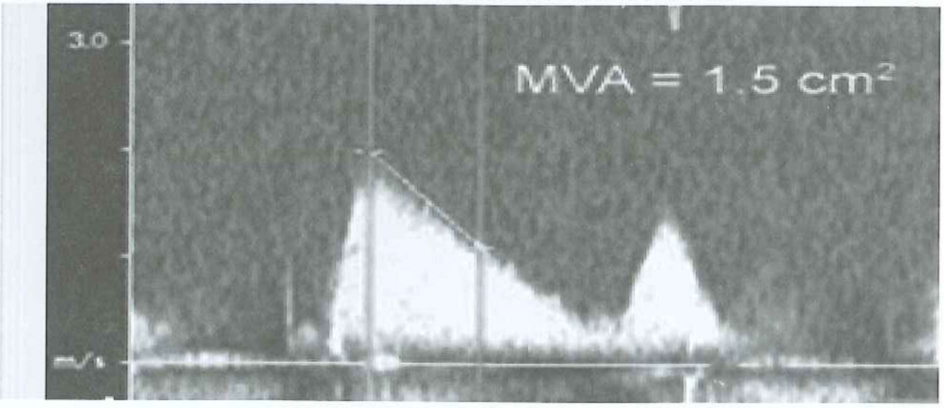


Diagram 51: showing calculated MVA by PHT method.

N.B: Regurgitation volume and regurgitation fraction are less commonly used in clinical practice, these calculations are discussed in " Hemodynamics"

Pitfalls in assessment of mitral stenosis:

I. By 2D echo:

A) Pitfalls related to the sonographer's technique:

1. Measuring the valve thickness using the tissue harmonic modality will over estimate the valve thickness.
2. Slanting cuts in the PSAX view will lead to overestimation of the planimetered mitral valve area.

So to overcome this, you must obtain a typical PSAX view. A typical PSAX is circular with the MV in the center of the view.

To identify the slating cut sections, the PSAX view will appear ovoid in shape rather than circular.(see before) .

B) B) Pitfalls related to the patient's clinical condition:

In rheumatic mitral stenosis, the most affected portion of the valve is the tips, while in calcification the base is more affected and the tips are usually spared. To overcome this pitfall, you must trace the mitral valve area at its tips and at its base. To do so, tilt the probe (very carefully and slowly) cranially from the PSAX view papillary muscle level until you see the mitral valve, if your motion was very fine then this portion of the mitral valve is supposed to be the tips, then by scrolling the track ball of the stored video you should planimeter the mitral valve area in the end diastole i.e. in a fully opened position. Then you must repeat your trace of the mitral valve (planimetry) at the base, to do so, you have to tilt the probe caudally (carefully and slowly) from a PSAX view great vessels level, if your tilting motion was very fine, then the first portion of the mitral valve seen is supposed to be the base, where the planimetry should be repeated as before. **The smallest valve area traced should be reported in a case of mitral stenosis.**

II. Mean P.G :

A) Pitfalls related to the sonographer's technique:

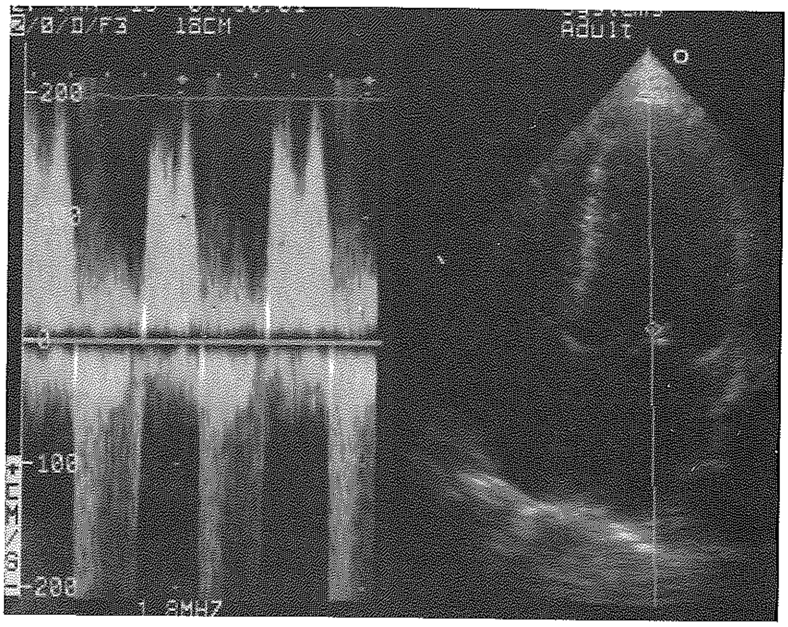
1. The angle between the U/S beam and the blood flow:

This angle will be referred to as angle theta "Θ"

As mentioned before, the angle of incidence between the U/S beam and the blood flow must be equal to zero. The calculated velocity will be underestimated proportionally by the value of the cosine of this angel of incidence. For example, in a case of mitral stenosis, the same case is illustrated in diagrams 52 (A) and (B). In diagram A, the angle of incidence is zero, notice that the measured velocity is 2 m/s (V=2m/s) , now look at diagram B where there is an angle between the curser and the blood flow, notice that the angle is nearly 45 degree, therefore the measured velocity is falsely underestimated.

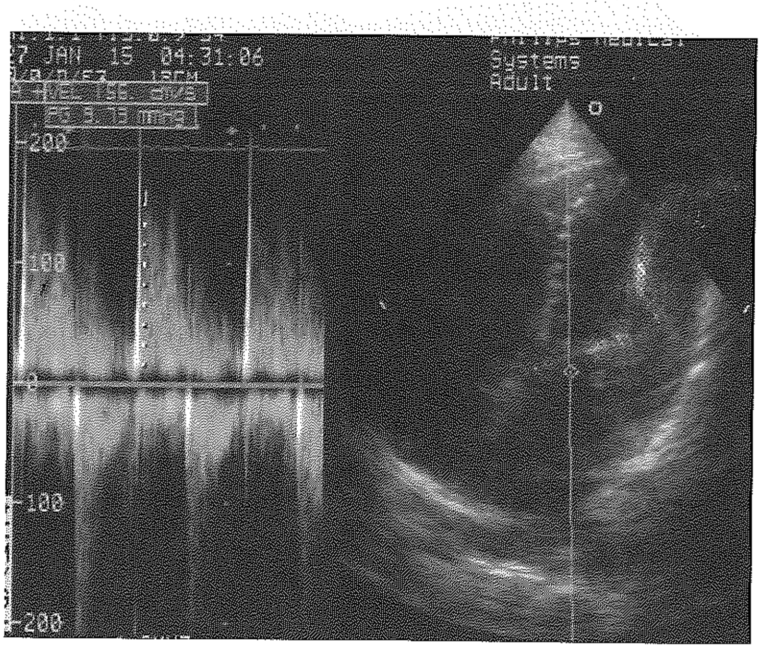
Diagram 52: the effect of cosine angle theta (angle Θ) on the velocity.

52 A:



Notice the typical Ap 4 ch view. With the curser line "U/S beam " aligned parallel to the blood flow from the LA to the LV, the velocity reached 2 m/s.

52 B:



Notice the angulated Ap 4 ch view. This will lead to an interception angle of about 45 degrees between the U/S Doppler beam and the blood flow from the LA to the LV. This will lead to false reduction in the calculated velocity. The velocity reaches only 1.5 m/s

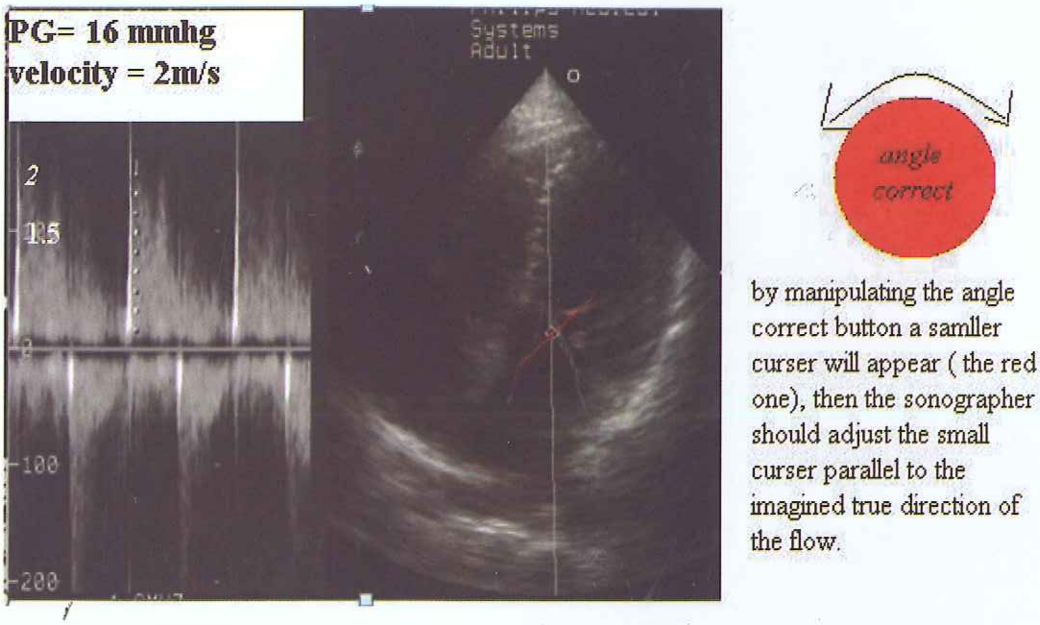
To overcome this pitfall, you must bring a typical apical 4 chamber or a 2 chamber view.

If the view is difficult to obtain, then you can apply an angle correct. The angle correct is a button that can be used in order to inform the machine about the angle of incidence. The machine calculates the cosine of this angle and automatically increases the measured velocities proportionally to the cosine value of the assumed angle. See diagram 53.

N.B the cosine angle Theta can only underestimate the measured velocities, but can never overestimate the measurements.

Diagram 53: Showing the manipulation of the angle correct button.

Notice that after adjusting the angle correct button (by rotating the button in most of the machine soft wares), the machine calculated the cosine of the "angle of incidence ϴ" and then automatically increased the calibration on the velocity axis proportional to the value of the cosine ϴ.



by manipulating the angle correct button a samller curser will appear (the red one), then the sonographer should adjust the small curser parallel to the imagined true direction of the flow.

B) Pitfalls related to the patient's clinical condition :

Any form of hyperdynamic circulation will lead to an increase in the velocity. Cases of hyperdynamic circulation includes three main circumstances: 1) any regurgitation lesion, 2) any tachycardia, and 3) any shunt lesions.

At low flow conditions, the velocity will be low. While in case of increasing the volume of the same fluid through the same orifice, the velocity will increase.

Typically, in a combined case of mitral regurgitation and mitral stenosis, the regurgitation volume will be added to the volume of the blood traversing the mitral valve in the subsequent beat, this will falsely increase the velocity detected by the Doppler study through the mitral valve.

The same concept applies for tachycardia, PDA and VSD.

N.B: There is no correction for this condition, and you must report the heart rate beside the calculated velocity, PPG or MPG.

N.B this is the same concept of the auscultated functional murmur (over flow murmur) in the same three cases.

III. Pressure half time :

A) Pitfalls related to the sonographer's technique:

1. Again, the angle of incidence between the U/S beam and the blood flow must be zero.
2. Extreme care must be taken to :
   a) Determine the peak velocity, i.e. the starting point from which the slope will decelerate.
   b) Accurately align the PHT line over the *bright line* of the deceleration slope.
   c) In a case of ski slope E wave, ignore the short steep earlier slope and use the flatter portion.

B) Pitfalls related to the patient's clinical condition:

1. Left ventricular compliance :

a) Poor relaxation i.e. grade I diastolic dysfunction.

Poor relaxation will lead to an overestimation of the PHT, leading to underestimation of the calculated mitral valve area. This is the same concept as blowing a balloon with poor compliant walls. The time needed for the pressure to fall to its half value will be prolonged.

In this case, it is very easy to differentiate weather the small calculated mitral valve area by PHT is either due to actual mitral stenosis or LV dysfunction. This will be achieved on the basis of the 2D echo appearance of the MV and by comparison to the planimetered area.

b) Non-compliant Left ventricle : i.e. grade III diastolic dysfunction

A non-compliant left ventricle will lead to an abrupt increase in the pressure followed by a rapid decrease. This will lead to a shortened PHT (the same physical concept as in the wide pulse pressure in a case of atherosclerosis, were there is no compliance to accommodate for the increasing pressure and also no compliance to maintain the pressure).

A shortened PHT will lead to a false increase in the calculated MVA.

To overcome this pitfall you must first diagnose a grade III dysfunction (see later) and in this case planimetry is more valuable than PHT derived MVA.

2. Left ventricular end diastolic pressure:

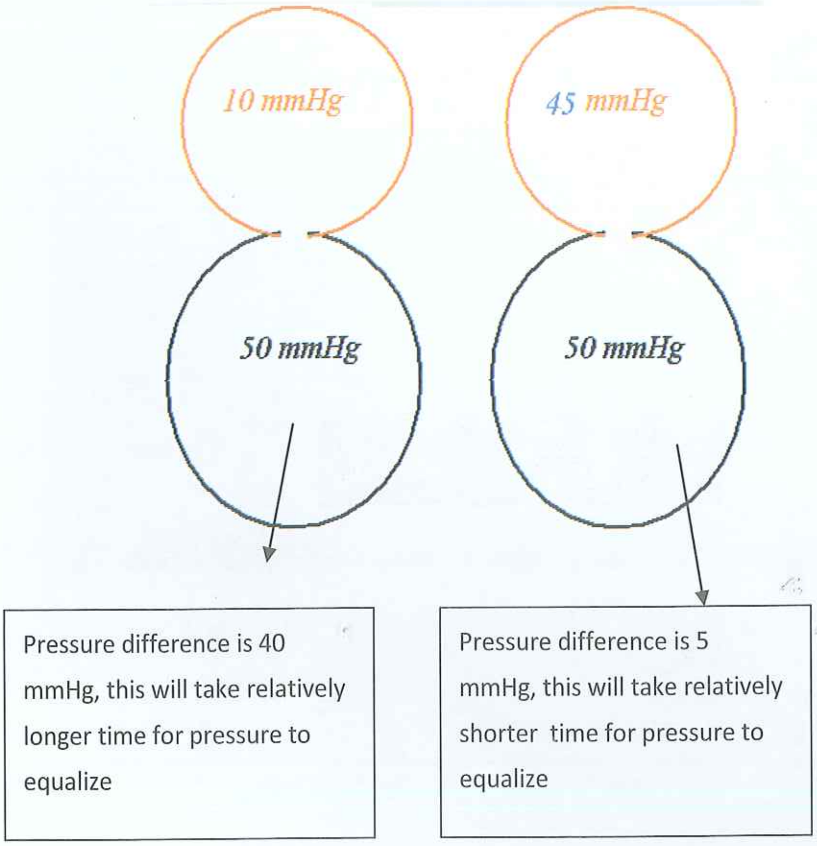
Elevated L.V end diastolic pressure: the most common causes are aortic regurgitation and end stage heart failure.

The physical concept is that the time needed for the pressure to fall to its half value will shorten if there is already an increased pressure in the other chamber. See diagram 54.

Also the increased LV pressure will lead to an early closure of the mitral valve that will " for sure" shorten the PHT.

To overcome this pitfall you must be aware of the conditions that increase the L.V pressure, and in this case the planimetry is more valuable than the PHT-derived MV area.

Diagram 54: The time needed for the pressure to fall to its half value will shorten if the pressure is elevated in the other chamber and vice versa.



Pressure difference is 40 mmHg, this will take relatively longer time for pressure to equalize

Pressure difference is 5 mmHg, this will take relatively shorter time for pressure to equalize

Summary of pitfalls:

Before using PPG think of dynamics.

Before using pressure half-time think of compliance and LV pressure.

Before using planimetery think of rheumatic heart disease.

Aortic stenosis:

Normal aortic valve, see diagram 55:

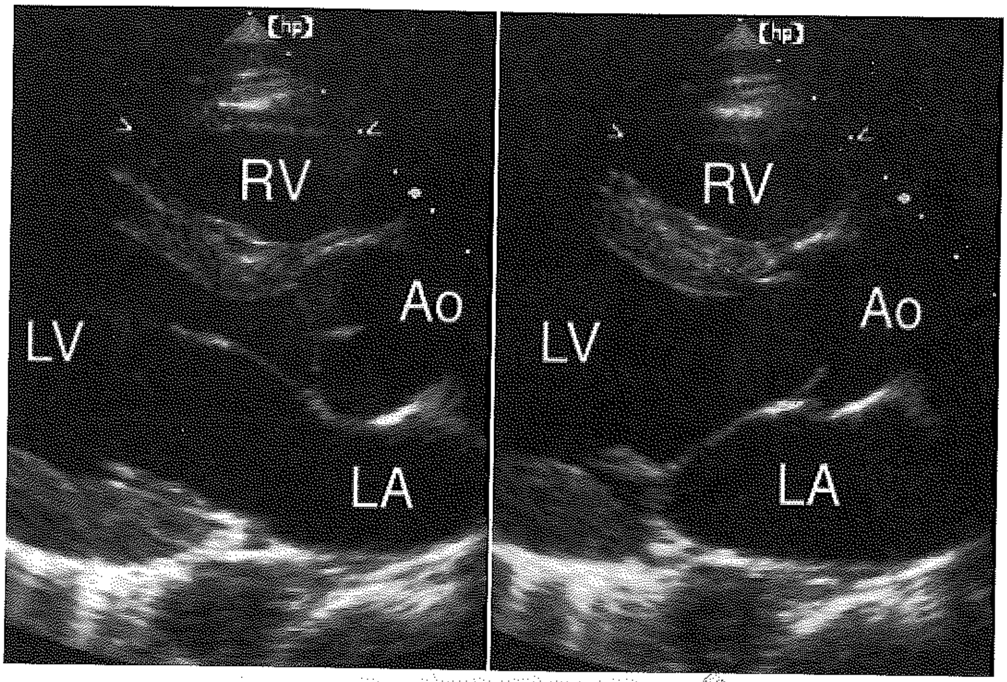


Diagram 55: Notice the normally thin aortic valve cusps, sometimes the cusps are normally difficult to image in the open position.

Aortic stenosis:

Causes of aortic valve stenosis :

Valvular, sub-valvular or supra-valvular.

However for each cause there will be prominent 2 D criteria. And PW must be used to localize the level of obstruction.

1) In valvular causes :

There will be overwhelming valve abnormality suggesting the cause.

2) Subvalvular obstruction:

This will fall in two main categories:

   a) Dynamic obstruction : HOCM, dehydration, end stage severe aortic stenosis, rare complication of myocardial infarction, post mitral valve replacement.
   b) Fixed obstruction : sub- aortic membrane.

3) Supra valvular obstruction :
   a) Sever forms of familial hypercholesterilimia.

b) Williams's syndrome.

2D echo: Thickened leaflets, restricted motion, areas of calcifications and sometimes rheumatic nodules may be seen. Diagram 56.

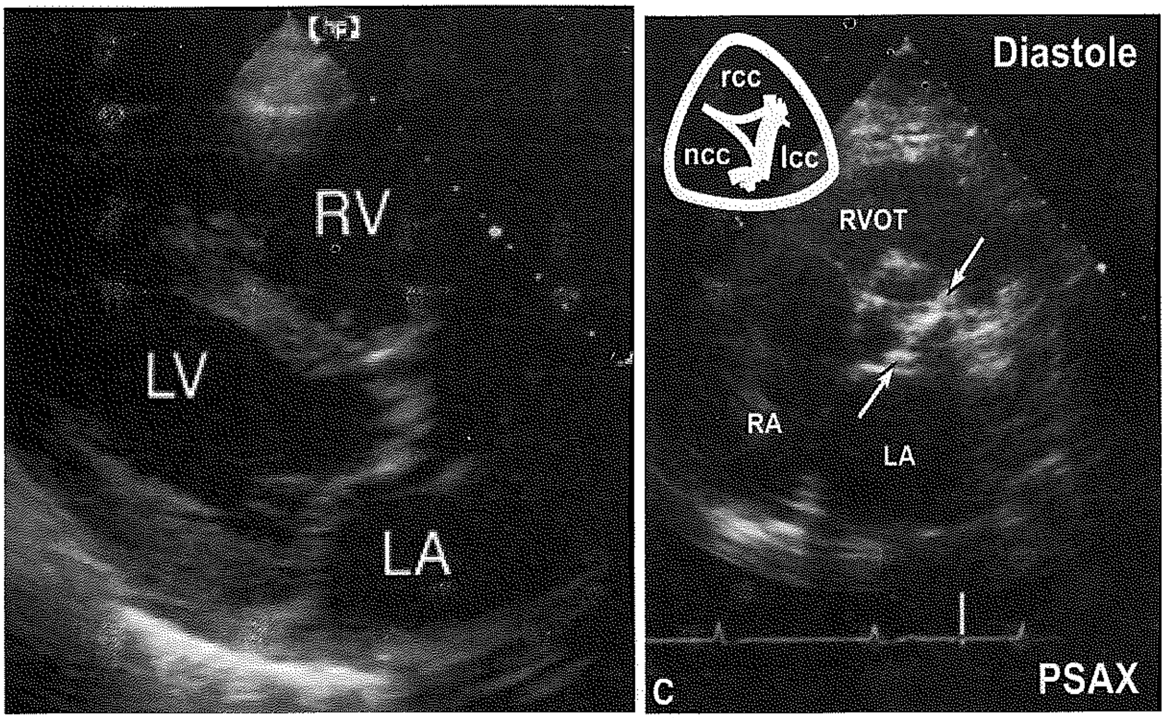
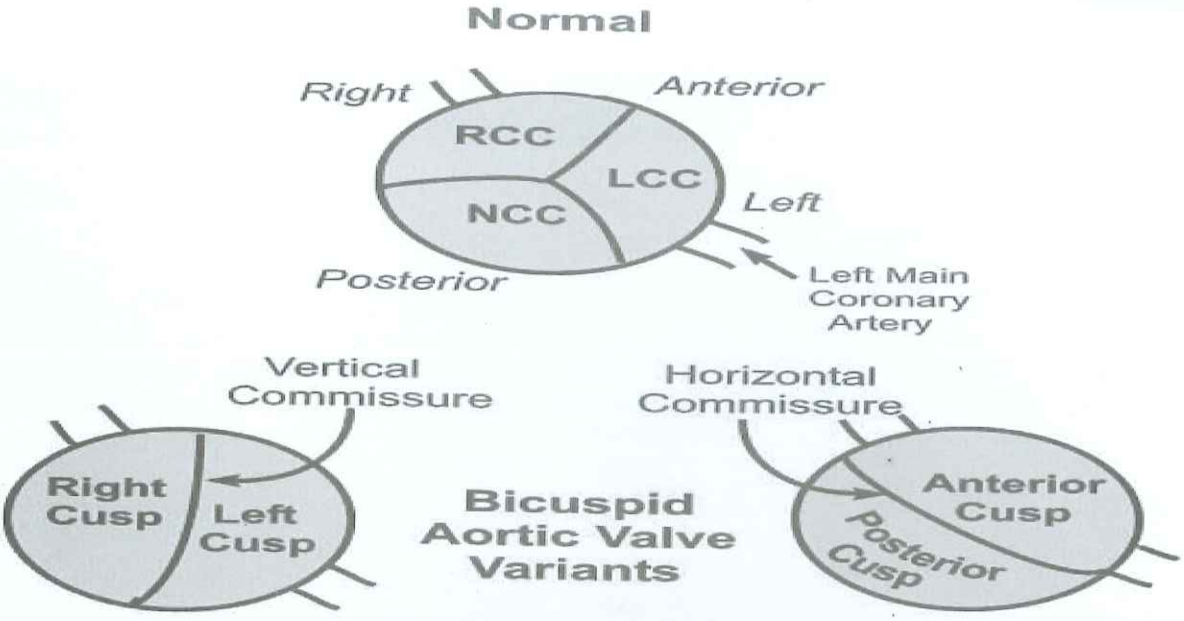


Diagram 56 Notice the calcific aortic valve (compare to the normal valve)

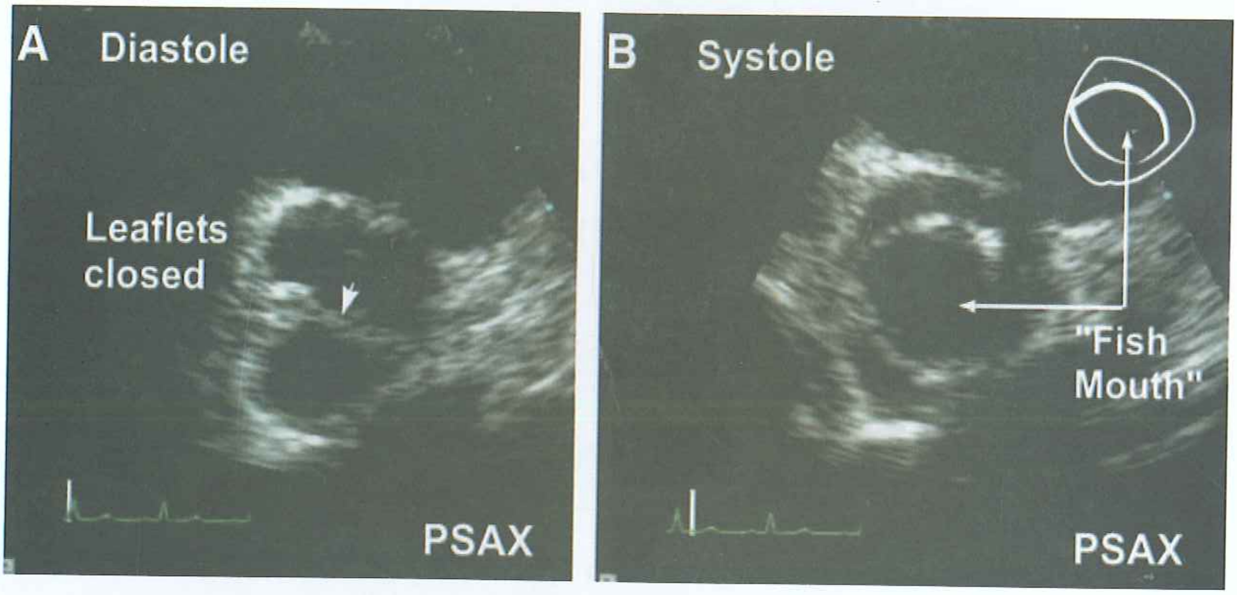
Care should be taken to judge whether the aortic valve is bicuspid or normal trileaflet aortic valve.

In a case of bicuspid aortic valve, other associations must be searched for namely: 1) dilated aortic root &/or aortic dissection , 2) Coarctation of the aorta.

The planimetry of the AV in the PSAX view aortic valve level correlates poorly with the actual valve area. However, if the aortic valve is clearly optimized, planimetry may be done. By using this technique, the actual valve area may be larger than the traced area, but never smaller. The planimetry of the aortic valve is better traced using TEE (mid esophageal level 55 degree aortic valve short axis view).



Bicuspid aortic valve variants



M.mode:

Restricted box shape opening of the aortic valve (decreased cusp excursion), this method is of little value in quantification of the aortic stenosis. Diagram 57 A : aortic stenosis. B : normal aortic valve.

Aortic stenosis

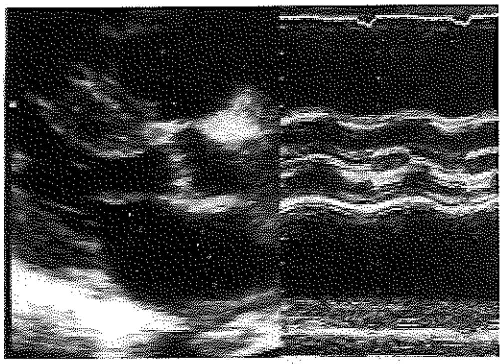


Diagram 57(A) Notice the restricted box shape and the thickened cusps

normal aortic valve.

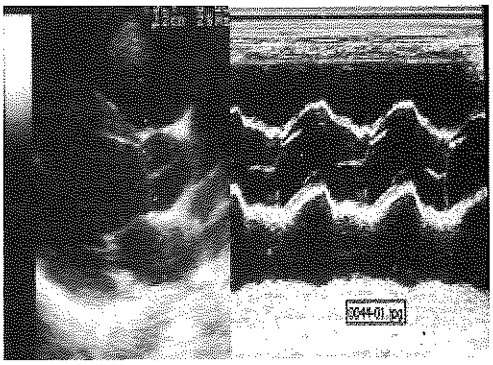


Diagram 57 (B) Notice the thin cusps in the M.mode study

Doppler :

This is the main modality in quantification of the aortic valve stenosis.

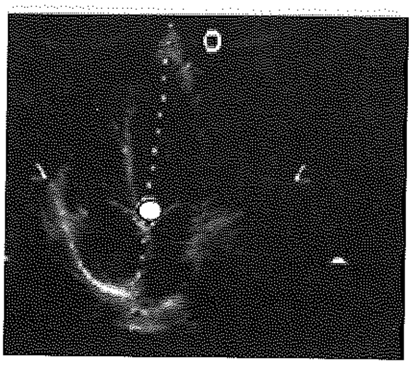
By aligning CW Doppler through the aortic valve in an apical view max velocity, PPG and MPG can be measured. (as in mitral valve, see before) See diagram 58.

See table for quantification of AV stenosis.

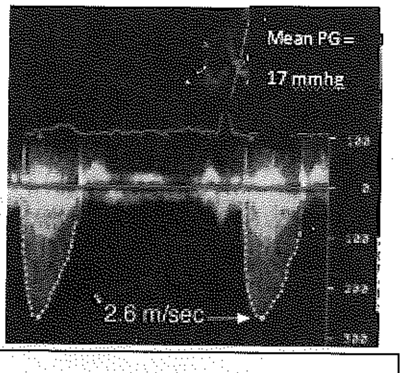
1) Use CW wave Doppler to search for high velocity jet.
2) If a high velocity jet is encountered, its origin must be localized by PW Doppler , either from the LVOT or from the aortic valve. See diagram 59 A & B.

1)

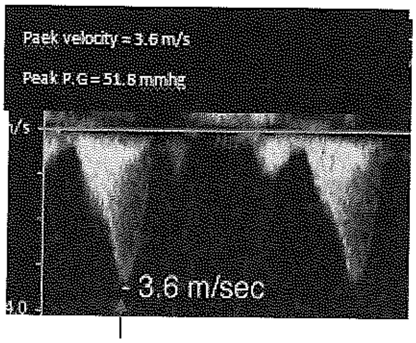
diagram 58: 1)a high velocity jet is encountered



CW Doppler is aligned over the aortic valve



CW Doppler shows a high velocity jet,
Mean PG is calculated

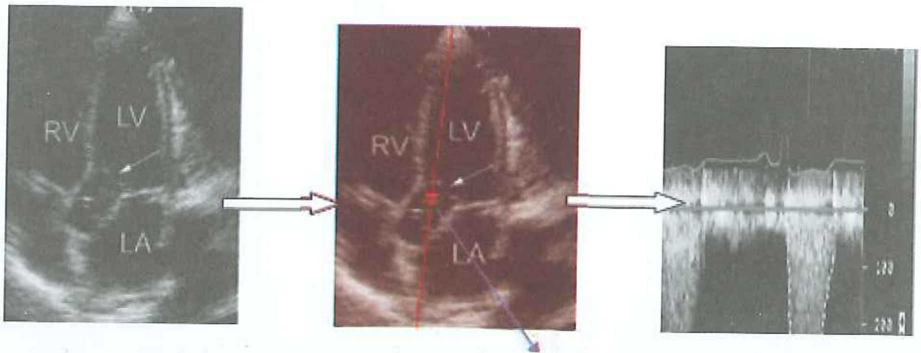


PPG is calculated too.

PW is fundamental in assessment of aortic stenosis. PW Doppler must be used to localize the high velocity jet whether valvular or subvalvular. In subvalvular stenosis the PW will show aliasing in the LVOT, while in valvular stenosis the PW will show normal velocity in the LVOT but an aliased velocity at the aortic valve. See diagram,

2) PW Doppler is used to localize the origin of the high velocity jet.

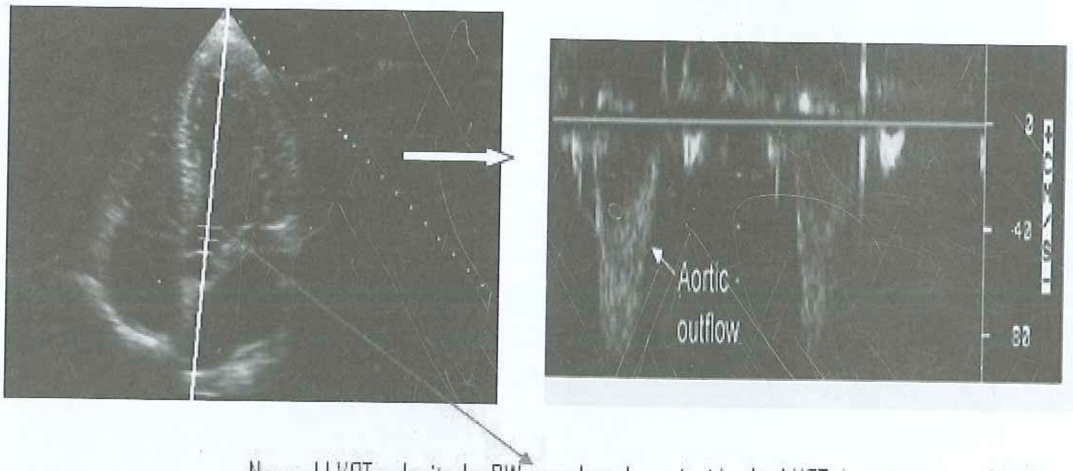
diagram 59 (A) : Example no 1: LVOT obstruction.



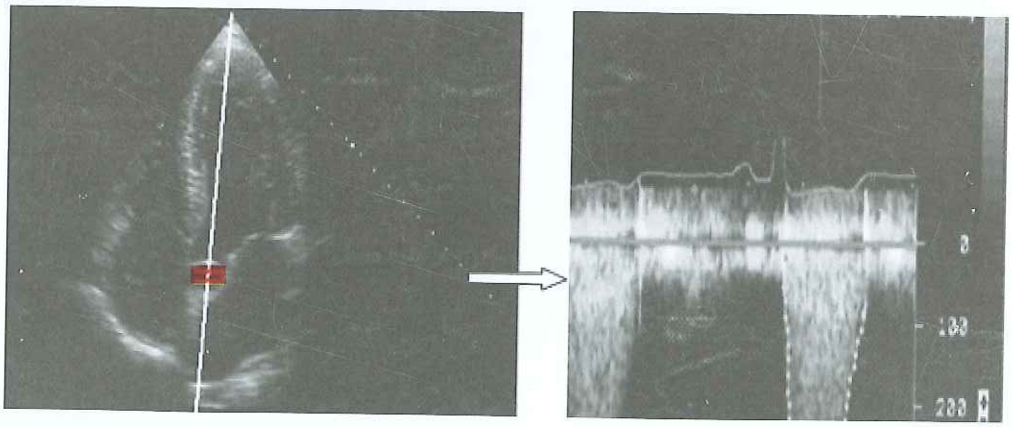
by 2 D echo a subvalvular membrane is shown, by using the PW with sample volume in the LVOT, an aliased velocity is found,

Denoting a high velocity jet originating from the LVOT i.e. subvalvular obstruction, in clinical practice a differential diagnosis must be suggested.

Diagram 59 (B) Example no.2 : valvular obstruction:



Normal LVOT velocity by PW sample volume inside the LVOT denoting normal LVOT.



Aliased velocity by PW at the aortic valve denoting valvular stenosis

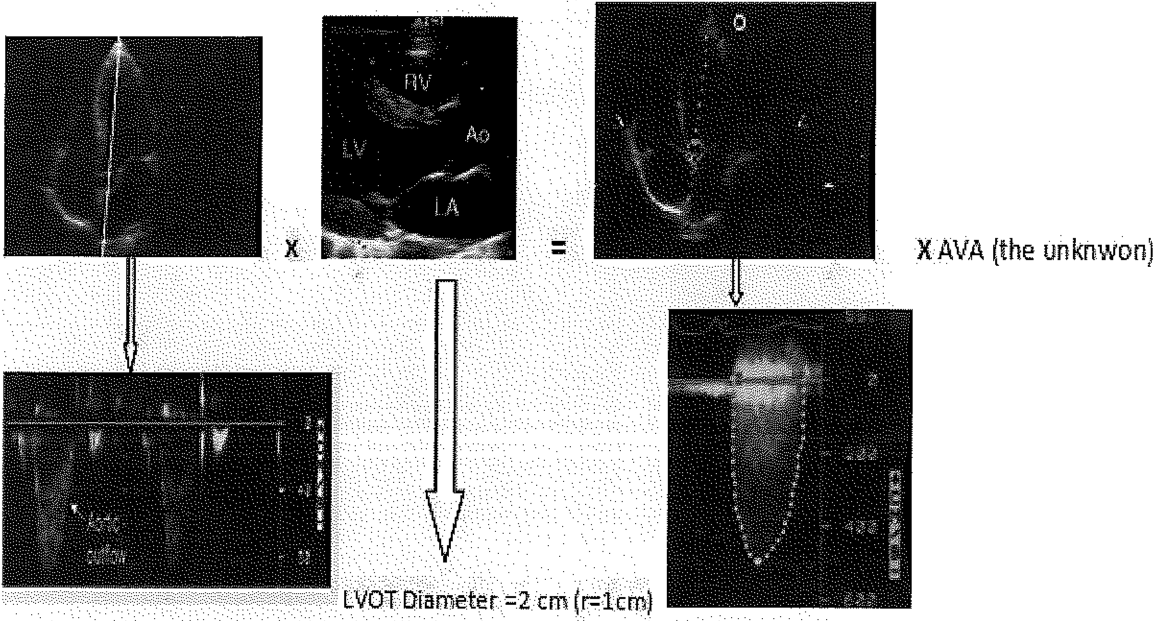
Continuity equation:

Another method is the continuity equation for evaluation of the aortic valve area Recall from the chapter of heamodynamics that the volume of blood traversing the LVOT equals the volume of the blood traversing the aortic valve, i.e. the stroke volume equals the blood traversing the aortic valve. See equation illustrated in the diagram 60.

Remember that to localize a velocity at a certain point we will use the PW, so at the LVOT precise location PW will be used. But the CW can be used to illicit the highest velocity along the curser line, this velocity is in fact within the AV, so CW will be used to illicit the aortic valve velocity.

Diagram 60:

LVOT VTI (PW) X LVOT area (Pi r^2) = Aortic valve VTI (CW) X Aortic valve area (AVA)



Continuity equation is validated in the presence of poor EF, and in the presence of AR.

Color Doppler:

Color Doppler will show a high velocity jet traversing the aortic valve, with proximal convergence zone towards the LV side and the high velocity jet across the aortic valve and into the ascending aorta. Color Doppler is not used in practice to quantify aortic stenosis. See diagram 61 A & B..

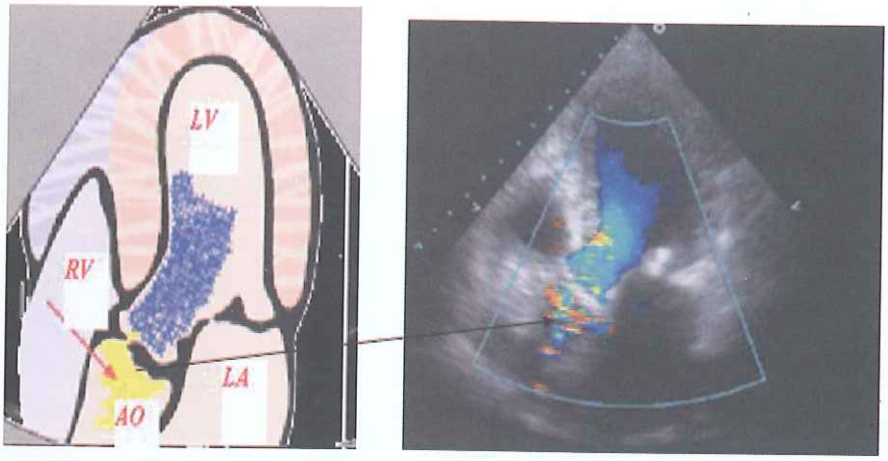


Diagram 61 : A high velocity jet demonstrated as a mosaic pattern by color Doppler in a case of aortic stenosis, notice the normal color variance inside the LVOT and the mosaic pattern after the valve.

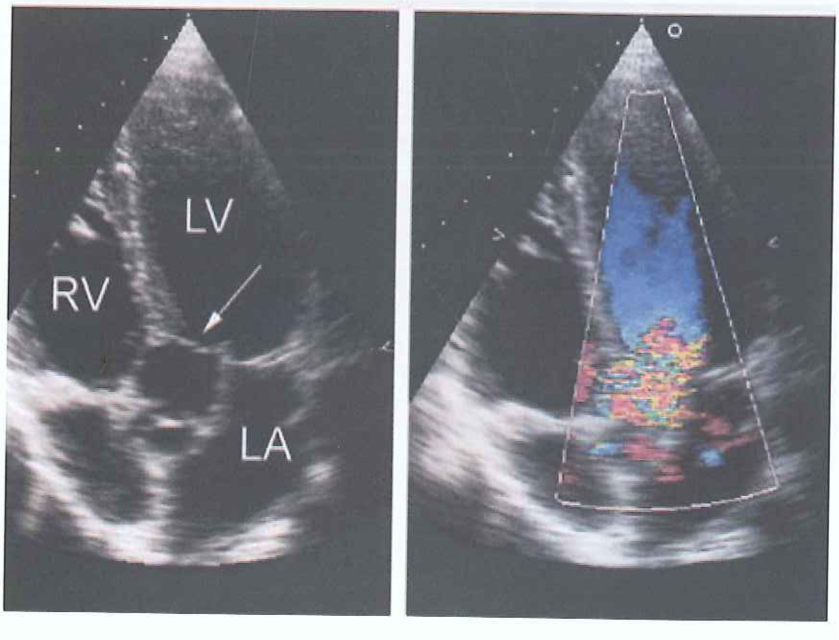


Diagram 61 B : A high velocity mosaic pattern is seen in the LVOT, denoting LVOT stenosis by color Doppler.

Grading the severity of aortic stenosis " A.S" by mean PG and peak PG (in absence of hyper dynamic circulation) and by Aortic valve area.

Normal	Mild A.S	Moderate A.S	Severe A.S
MPG : < 8 mmHg	8-20 mmHg	20-40	>40 mmHg
PPG : 15 mm Hg	15- 35 mmHg	35-65 mmHg	>65 mmHg
Area : 3-4 cm^2	1.5 -2cm^2	1-1.5cm^2	<1 cm^2

N.B : in the presence of any motion restriction of the aortic valve plus a velocity of more than 1 m/s , aortic stenosis is diagnosed even if the PPG and MPG ranges lie within normal ranges

In sclerocalcific valves the aortic valves should have normal excursion and the PPG should be less than the mild range.

Other less commonly used equations to determine aortic stenosis:

Stroke work loss = 100 X MPG aortic valve / MPG aortic valve + SBP

SBP : systolic blood pressure

Normally it is supposed to be a low value.

Cut off value of more than 25 % is used to identify patients with aortic stenosis and poor survival outcome.

Pitfalls in assessment of aortic stenosis :

By 2D echo :

1) Patients with poor L.V function will have poor excursion of the AV opening. This may be mistaken at the first glance for aortic stenosis, but, the absence of thickness and calcification and the presence of poor L.V function easily clear out this situation. For further identification of a case of pseudo aortic stenosis, stress echo may be indicated (see later).
2) Care should be taken to identify bicuspid valves. Diagram 62

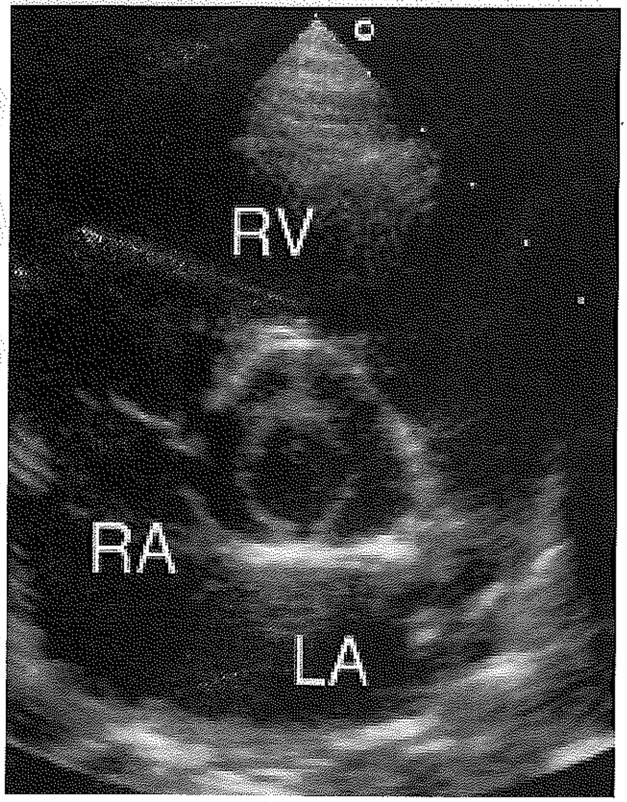


diagram 62 : bicuspid aortic valve.

By M.mode : The same as 2D echo

Eccentric closure line indicates bicuspid valve . See diagram 63.

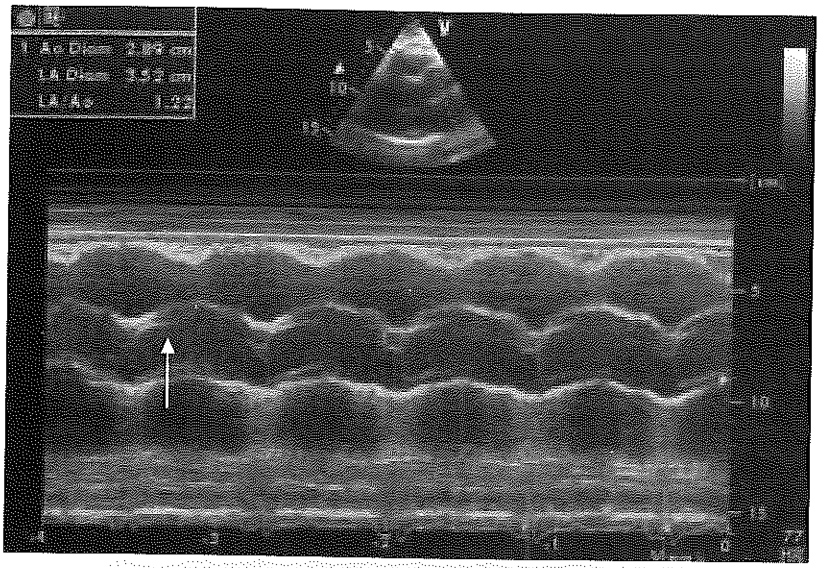


Diagram 63 : bicuspid Aortic valve (M.mode trace), notice the eccentric closure line denoted by the arrow.

By Doppler :

Pitfalls due to poor sonographer's technique:

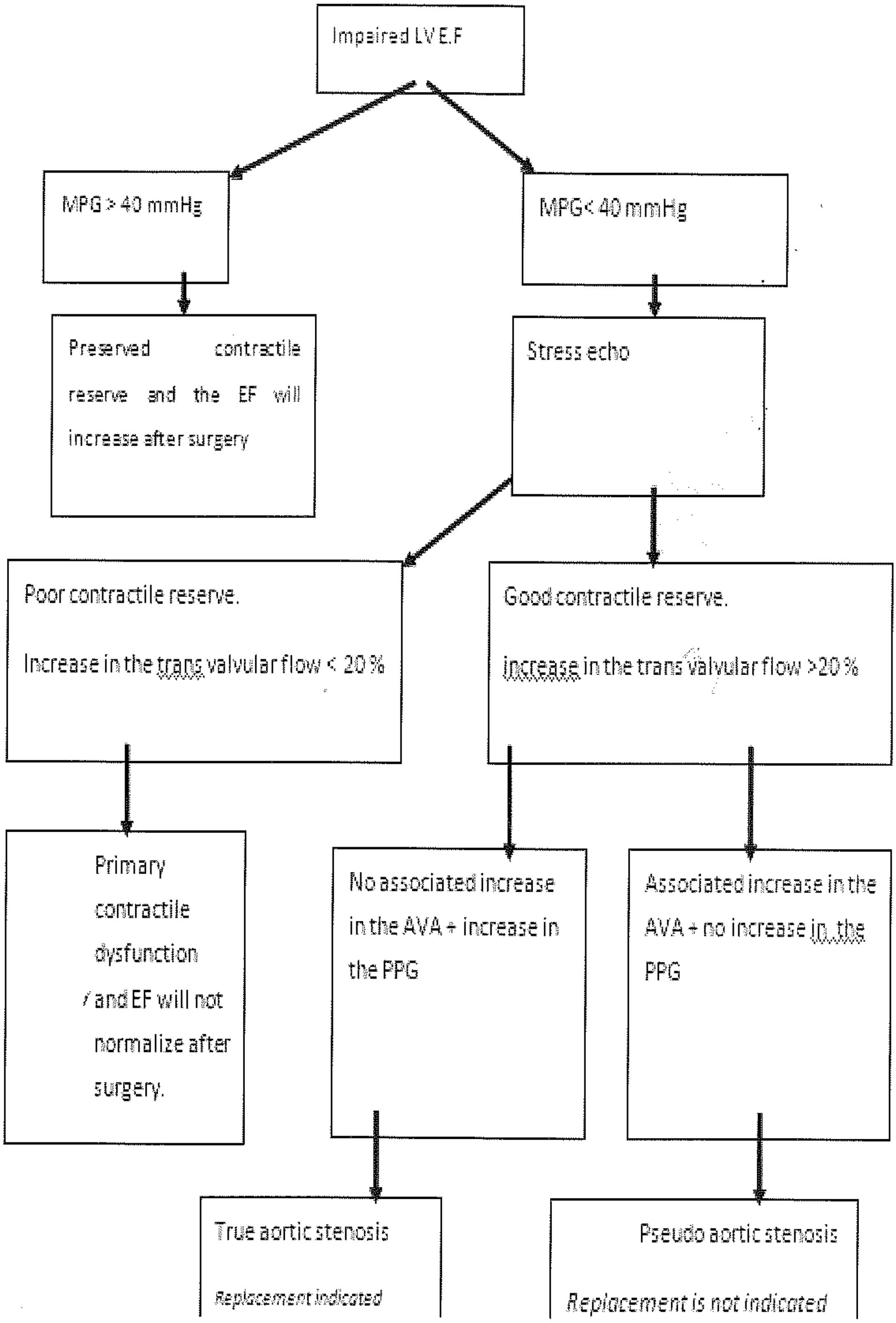
1) As usual, the angle of incidence "theta" between the U/S beam and the blood flow, must be nearly zero, i.e. the curser is parallel to the blood flow. Any angulation will decrease the elicited velocity proportional to the cosine value of this angle.
2) Any case of hyper dynamic circulation will lead to overestimation of the pressure gradients. In this case the continuity equation can be used.

Pitfalls due to patient's clinical condition:

clinical situations that may be seen in a patient with aortic stenosis and may lead to fallacies in judgment of the severity of the aortic valve stenosis:

1) **Poor ejection fraction:**
   In a patient with a poor ejection fraction the peak pressure gradient will be underestimated. This should be differentiated whether this is due to a primary cardiomyopathy or secondary to the valve lesion. In primary cardiomyopathy the patient will not benefit from aortic valve replacement and the decreased cardiac function will continue even after valve replacement. In secondary cardiomyopathy due to the valvular obstruction, the ejection fraction is likely to increase after the valve replacement if the patient still has a cardiac reserve as should be demonstrated by stress echo prior to the valve replacement. See algorithm.

EMSE clinical insights from the guidelines for aortic valve low flow low gradient and in patients with impaired LV function:


Impaired LV E.F
MPG > 40 mmHg
MPG< 40 mmHg
Preserved contractile reserve and the EF will increase after surgery
Stress echo
Poor contractile reserve.
Increase in the trans valvular flow < 20 %
Good contractile reserve.
increase in the trans valvular flow >20 %
Primary contractile dysfunction and EF will not normalize after surgery.
No associated increase in the AVA + increase in the PPG
Associated increase in the AVA + no increase in the PPG
True aortic stenosis
Replacement indicated
Pseudo aortic stenosis
Replacement is not indicated

N.B: In low flow AS, the Grolin formula underestimates the aortic valve area.

2) **Aortic pseudo stenosis:**

In a patient with poor ejection fraction, the aortic valve excursion will be limited and may mimic aortic stenosis. Differentiation of pseudo aortic stenosis from anatomical aortic stenosis will be done on basis of stress echo. Also a calcified and thickened aortic valve clearly suggests a primary valve problem. See diagram 64.

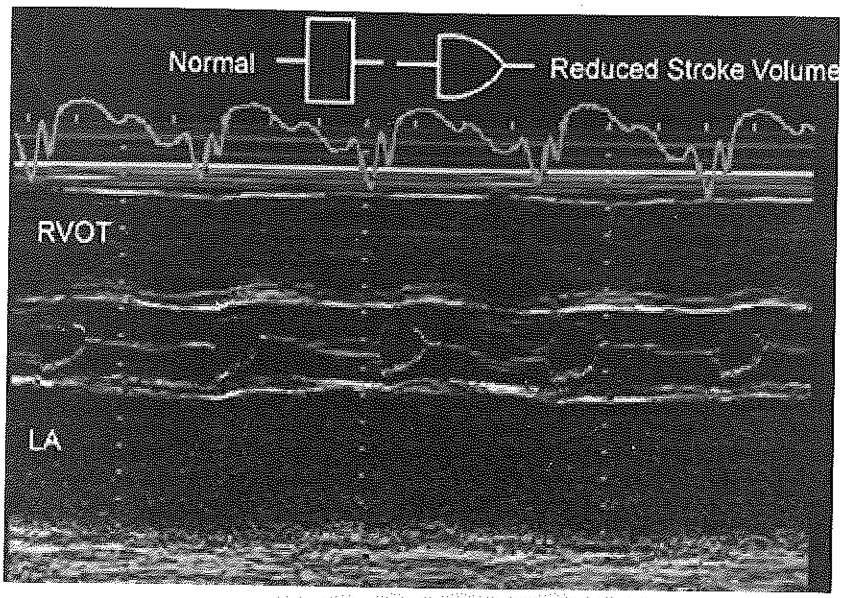


Diagram 64 : Poor excursion of the aortic valve as shown by M.mode.

3) **Mitral stenosis:**

Mitral stenosis will decrease the forward flow over the aortic valve leading to a reduction in the pressure gradient. Performing a balloon mitral valvoplasty to the stenotic mitral valve will generate pressure gradient over the aortic valve. So an upper border line moderate aortic stenosis will turn to be a severe case of aortic stenosis once the mitral stenosis is relieved.

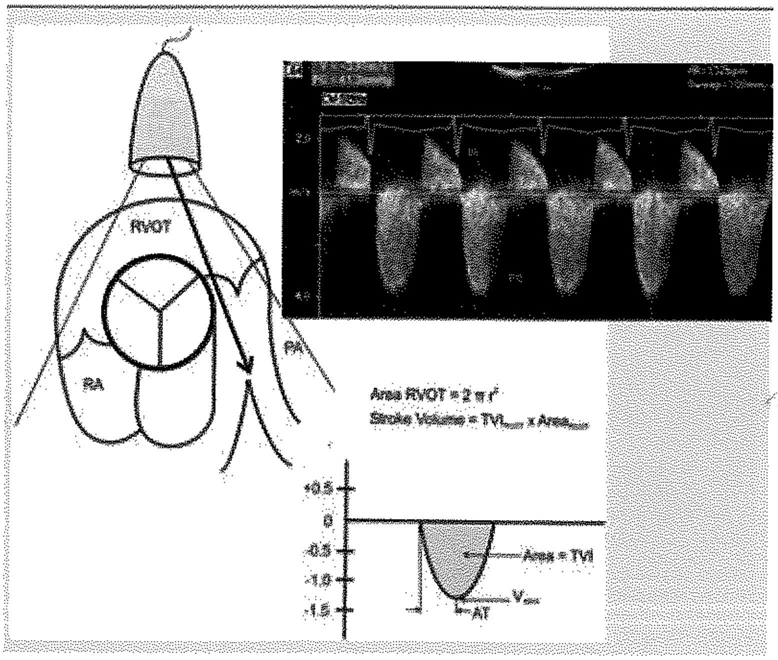
4) **PW Doppler must be used to localize the level of the stenosis either subvalvular or valvular (see before)**

Confusion between mitral regurgitation and aortic stenosis signals may arise in cases of anteriorly directed mitral regurgitation, where the continuous wave aligned over the aortic valve may be contaminated by the mitral regurgitation jet. Mitral regurgitation jet should not be confused with aortic stenosis; the mitral regurgitation signal is parabolic and always of high velocity, while aortic flow is triangular in shape (or dagger-shaped in cases of HOCM) and the velocity will differ according to the severity of the lesion. Also confirmation by PW Doppler is mandatory to prove that the high velocity jet originates from the aortic valve itself (aliasing will occur when the PW sample volume is placed at the aortic valve).

Pulmonary stenosis "PS":

The same concepts used to asses AS will be used to assess PS, i.e. 2D imaging, peak velocities, PPG and MPG. This can be done in any view showing the pulmonary valve. See the example in diagram 65. According to the guidelines, the decisions to intervene in a case of pulmonary stenosis will be made upon the invasive pressures derived by right heart catheterization.

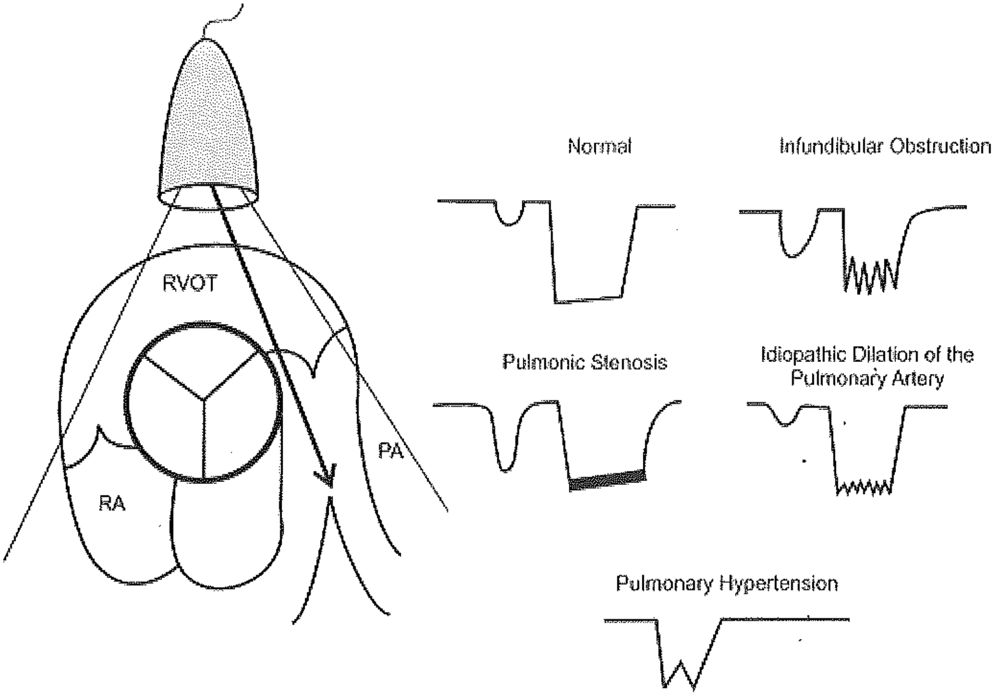
Diagram 65: showing pulmonary stenosis, denoted by the high velocity –ve deflection signal. N.B the +ve deflection signal refers to an associated pulmonary regurgitation.



2D imaging may show a hypoplastic pulmonary valve, hypoplastic RVOT or even a hypoplastic main pulmonary artery.

M.mode :

The motion mode is of little value in the assessment of PS see below.



Schematic representation of M-mode echocardiograms of normal and abnormal pulmonary valves. In the normal schematic, note the normal A-wave and box-like opening of the valve. Various disease states are also schematized

M.mode traces in various pathologies that may affect the pulmonary flow and pressures.

Doppler study:

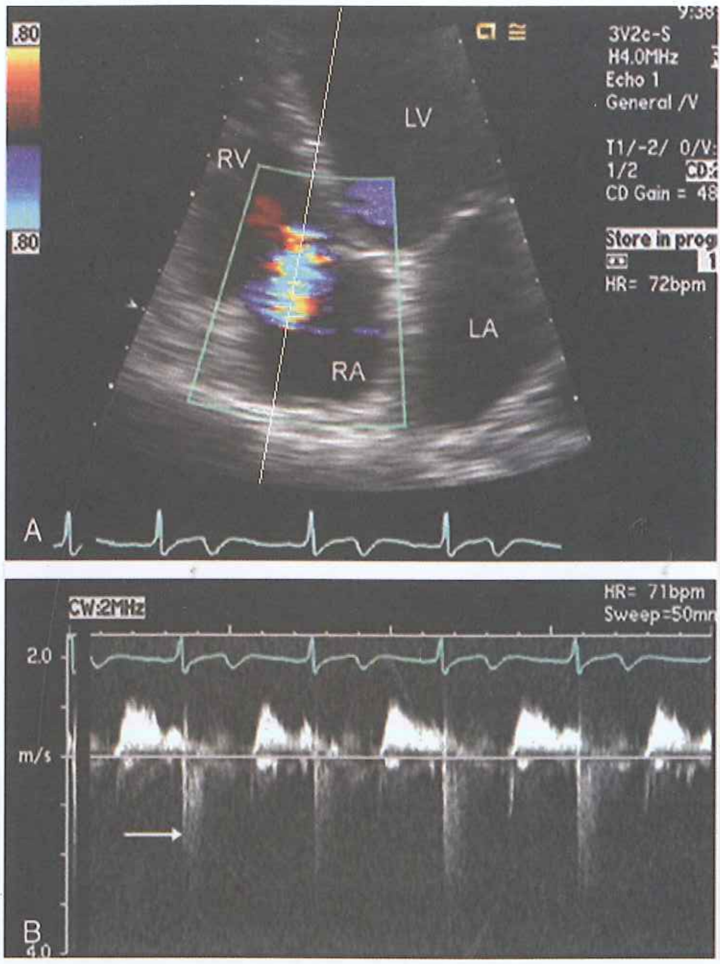
This is the main modality used to asses PS

The same concepts used to asses AS will be used to asses PS. See diagram 65.

Tricuspid stenosis "TS":

The same concepts used in mitral stenosis will be used to assess the tricuspid valve velocities, PPG and MPG. This can be done in any view showing the tricuspid valve. See an example in diagram 66. N.B: Tricuspid valve area = 210/ PHT of the tricuspid valve (less commonly used).

Diagram 66: Showing an example of tricuspid stenosis. Notice the high velocity TV velocity.



- 2D imaging may show a calcified TV, if due to rheumatic activity the mitral valve will almost always be affected. If the mitral valve is free, this should raise the suspicion towards carcinoid syndrome that typically affect the right sided valves and spares the left sided ones. (the active metabolite "serotonin" that cause direct valvular damage is deactivated in the lung)
- M.mode : little value
- Doppler study: exactly the same as in mitral stenosis.
  Severe TS: MPG>5 mmHg, mild TS: MPG < 5 mmHg

Chapter 7: Regurgitation lesions:

The main modality to quantify the regurgitation lesions is the color Doppler, mainly to calculate the percentage of the regurgitation jet from the chamber, and measure the vena contracta.

Recall from "clinically applied physics", that color Doppler mainly encodes the blood flow in terms of velocity, however, it was found that it also correlates with the amount of the blood flow (in absence of pitfalls).

Mitral regurgitation:

Causes of mitral regurgitation:

LEAFLET ABNORMALITIES

- Myxomatous degeneration of leaflets with excessive motion (most common)
- Rheumatic disease: scarring and contraction lead to loss of leaflet tissue
- Endocarditis: can cause leaflet perforations and retraction in healing phase
- Aneurysms: usually from aortic valve endocarditis; aortic insufficiency produces jet lesion on mitral valve
- Congenital:
  - Cleft mitral valve: isolated or with ostium primum atrial septal defect
  - Double-orifice mitral valve
- Hypertrophic cardiomyopathy: systolic anterior motion of the mitral valve

MITRAL ANNULAR ABNORMALITIES

- Annular dilation
  - From left ventricular dilation: dilated cardiomyopathy, ischemic disease, hypertension
  - Normal 10 cm in circumference
  - With sufficient dilation, loss of adequate leaflet coaptation
  - Tethering of leaflet and chordae can occur and produce relative restriction of leaflet motion
- Mitral annular calcification
  - Degenerative disorder, most commonly seen in the elderly
  - Accelerated by hypertension or diabetes
  - Also seen in renal failure with dystrophic calcification
  - Also seen with rheumatic heart disease
  - Marfan's syndrome, Hurler's syndrome
  - Mitral regurgitation results from immobility of the annulus, loss of sphincter activity

CHORDAL ABNORMALITIES

- Chordal rupture (most severe form is flail leaflet) results in loss of leaflet support usually with myxomatous degeneration
- Rheumatic heart disease (chordal fibrosis and calcification)

PAPILLARY MUSCLE ABNORMALITIES

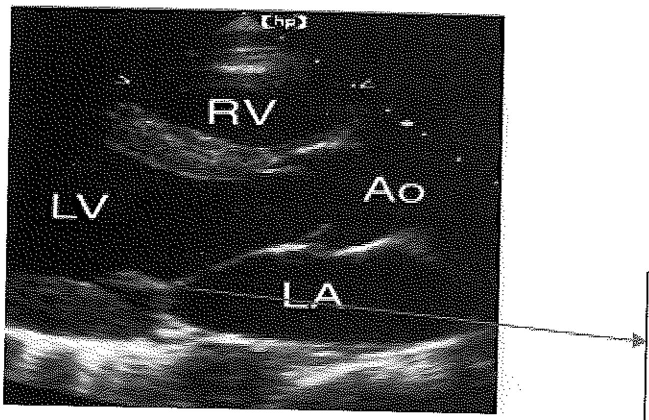
- Rupture with myocardial infarction
  - Complete rupture typically not survived
  - Partial rupture more typically encountered
- Dysfunctional papillary muscle
  - Ischemia
    - Posteromedial papillary muscle, single blood supply through posterior descending artery
    - Anterolateral papillary muscle, supplied by left anterior descending artery and left circumflex artery
  - Infiltrative processes: amyloid, sarcoid
  - Congenital: malposition, parachute mitral valve

Echocardiography findings:

2 D echo: (First remember what a normal mitral valve should look like, see diagram 67)

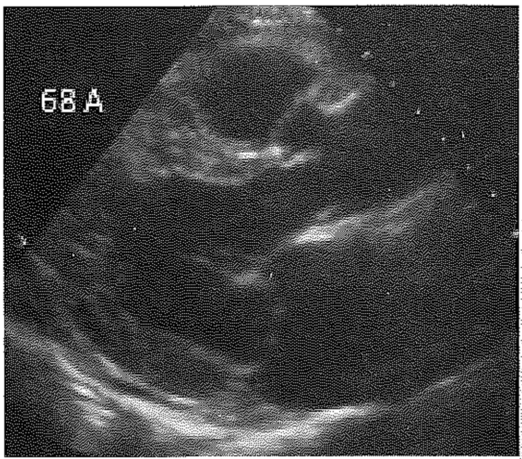
The cause of MR may be evident on 2D imaging, for example:

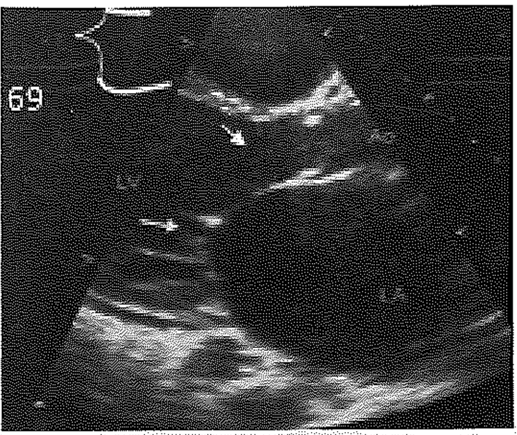
- A prolapsed MV may be clear from a PLAX view (diagrams 68 A&B): the leaflets of mitral valve in an apical 4 ch view, because in this view it may look normally as if prolapsing.
- Tethered MV may be evident in an AP4 chamber view or in a PLAX view diagram 69. Notice the poor cooptation zone (compare to the normal valve in diagram 67)
- Perforated mitral valve can be searched for, see diagram 70. Notice the loss of tissue within the mitral leaflets shown by the arrow.
- Flail mitral valve see diagram 71. Notice that the tips of the mitral valve are pointing towards the base of the heart, this could make an important trick to differentiate between severe cases of prolapsed mitral valve and flail valve, with the former having the tips pointing towards the apex of the heart.

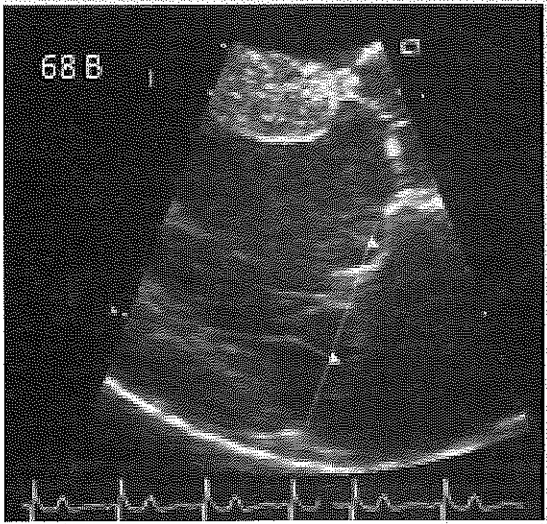


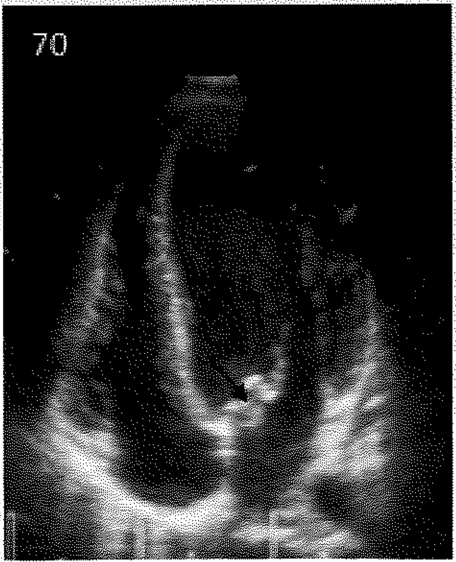
Notice the normal cooptation zone (zona coapta) in the form of 2mm of overlap between the two leaflets

Diagram 67 : normal mitral valve in 2 D echo


68 A


69


68 B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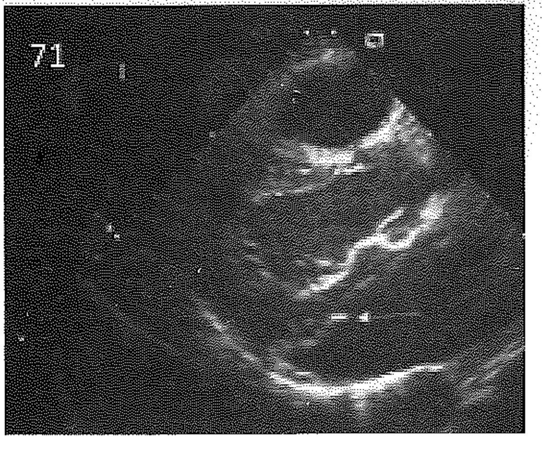
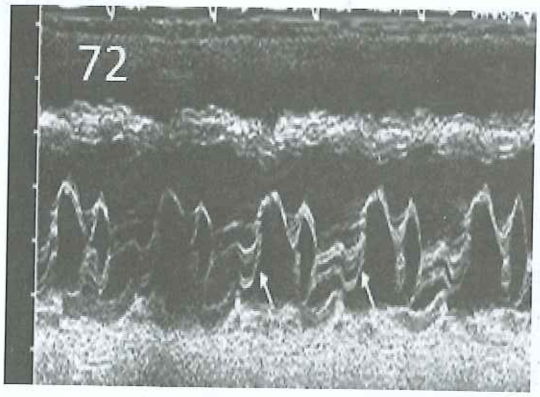
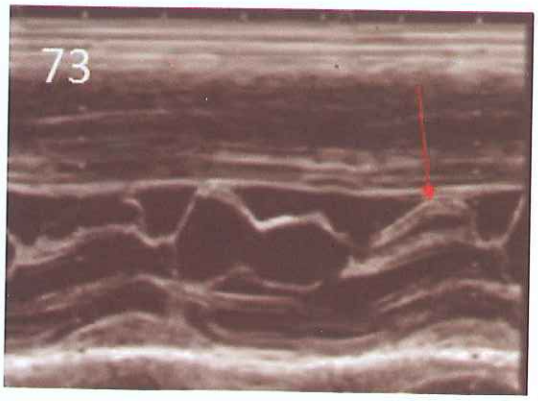

71

M.Mode:

A prolapsed CD line draws the attention for MVP, diagram 72. Notice the posterior saddling of the CD line denoted by the arrow.

Systolic anterior motion of the mitral valve (SAM) of the CD line also clarifies the cause of MR, and will be a part of dynamic LVOT obstruction, HOCM, or even HCM diagram 73. The SAM of the MV is pointed to by an arrow (see later).





Doppler :

CW Doppler will show a high velocity –ve signal 'U' shaped signal of MR. notice that MR must have a high velocity signal (LV pressure about = 120 mmHg, LA pressure about = 5 mmHg), so either a mild, moderate or severe MR will have a high velocity (it is a matter of PG between the atria and the ventricles) diagram 74.

Notice that the density of the flow correlates with the severity in a subjective manner.

PW Doppler has a little role in MR.

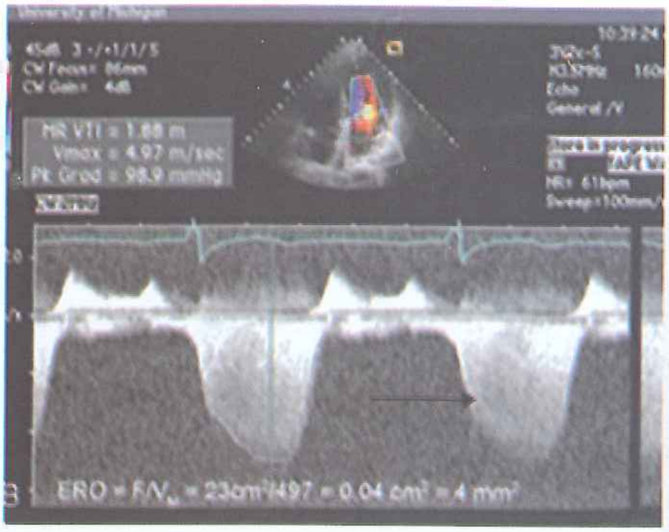


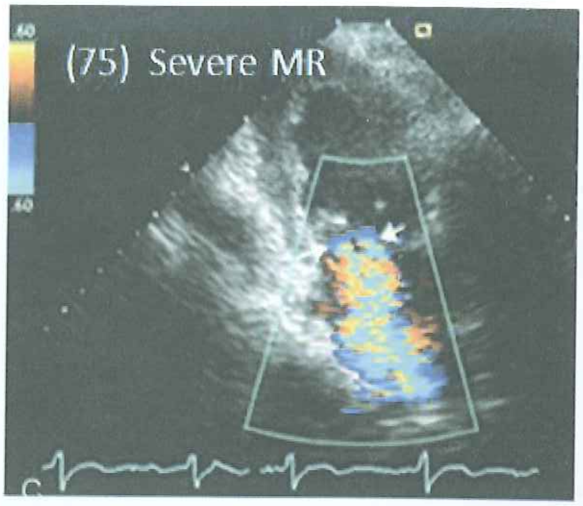
Diagram 74 : mitral regurgitation signal shown by the arrow.

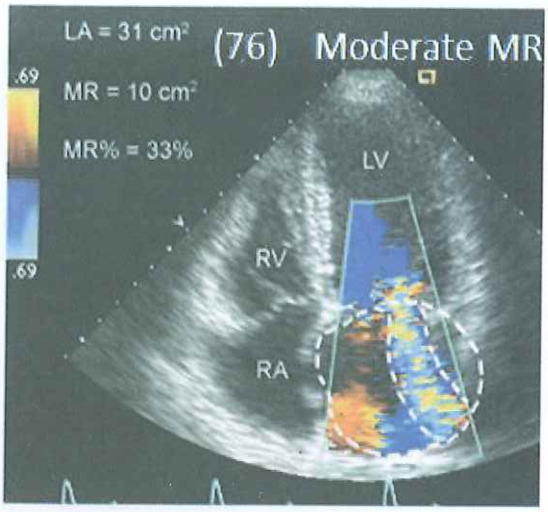
Color Doppler:

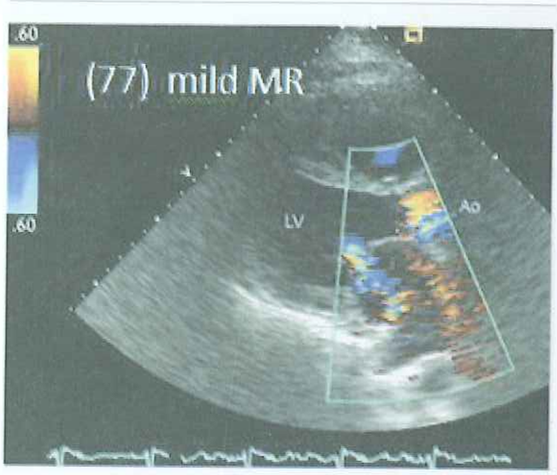
In the absence of pitfalls, the color Doppler correlates well with the amount of regurgitate blood. By aligning the color spectrum over the basal part of the LV, the MV and whole LA, the blue mosaic regurgitation jet can be elicited and traced. Remember that the mitral regurgitation jet will appear as a blue mosaic ellipsoid jet. The mosaic color is due to the high velocity jet owing to the big pressure difference between the LV and the LA during systole.

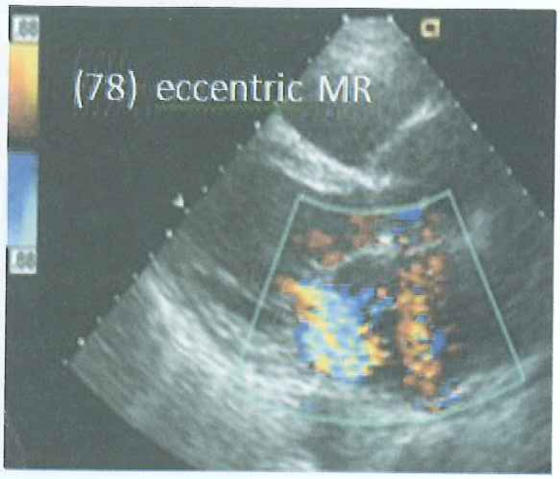
See diagrams 75, 76, 77, 78

For grading of severity see table below.









The **EMSE** describes the high velocity jet as being mostly ellipsoid in shape with well delineated borders, and lasts for the systolic time, i.e. not flashy. *A flashy non ellipsoid jet mostly will be a ghost artifact*.

Vena contracta :

It is defined by many authors as the narrowest part of the regurgitation jet just downstream the valve. As the regurgitation orifice increase the vena contracta will be wider. Diagram 79.

EMSE definition of vena contracta: The narrowest part of the regurgitation jet at the zona coapta just downstream the PISA.

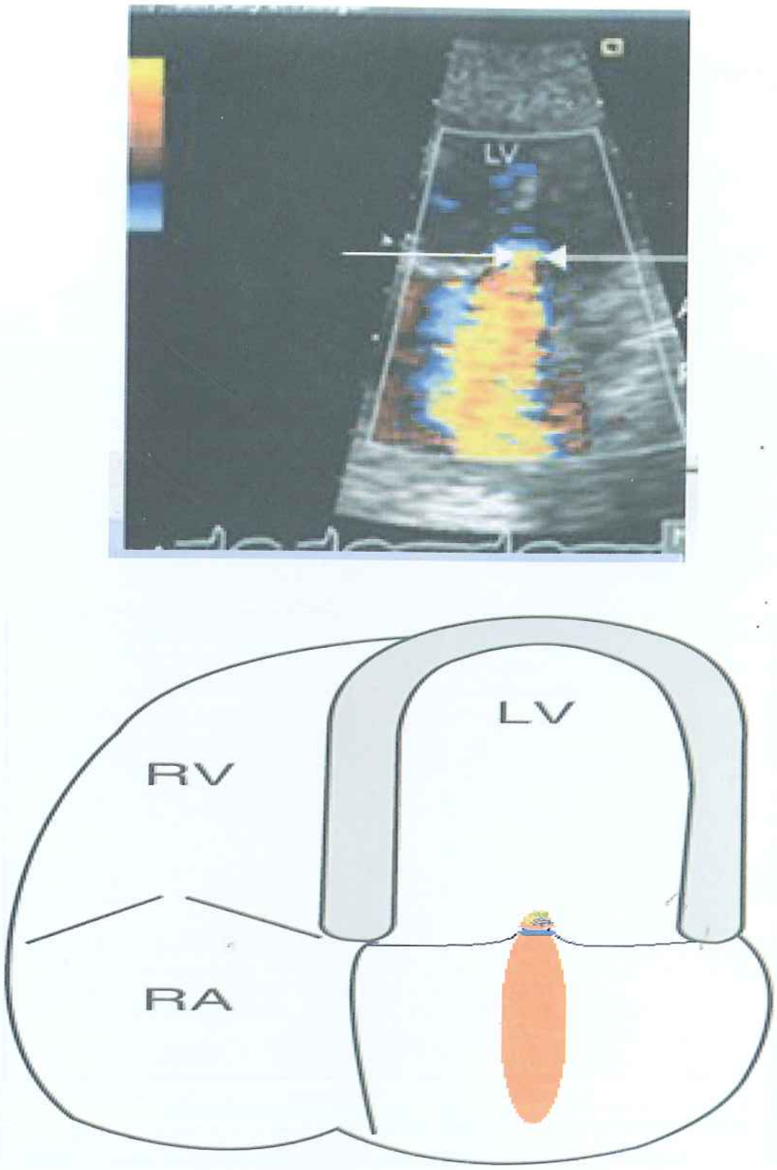


Diagram 79: the exact site to measure the vena contracta.

N.B:

1) Extreme care should be taken while measuring the vena contracta, as the measurement must be just below the cooptation point, this will appear as if the measurement is at the valvular level (true measure). But if the measurement is taken too much downstream the valve, it will lead to a huge overestimation of the vena contracta, and consequently overestimation of the regurgitation severity. See diagram 79.

2) In our school in the **Egyptian medical society of echocardiography "EMSE"**, we only trust vena contracta when it is a clear measure.

Grading of mitral regurgitation " MR" by color Doppler :

Mild MR grade I	Moderate grade II	Moderate grade III	Severe IV
Vena contracta < 3mm	3-6 mm	3-6mm	>6 mm
% of the LA <15 %	15- 30 %	30-50 %	>50 %

N.B in grade III & IV moderate to severe LA &/or LV dilatation is almost always present.

N.B: the PISA method to quantify regurgitation lesions is less commonly used in clinical practice and is not validated in MR associated with mitral valve prolapse.

In trauma, the posterior MV leaflet is more prone to tear than is the anterior leaflet.

Pitfalls in MR:

I. Pitfalls related to the patient's medical conditions :

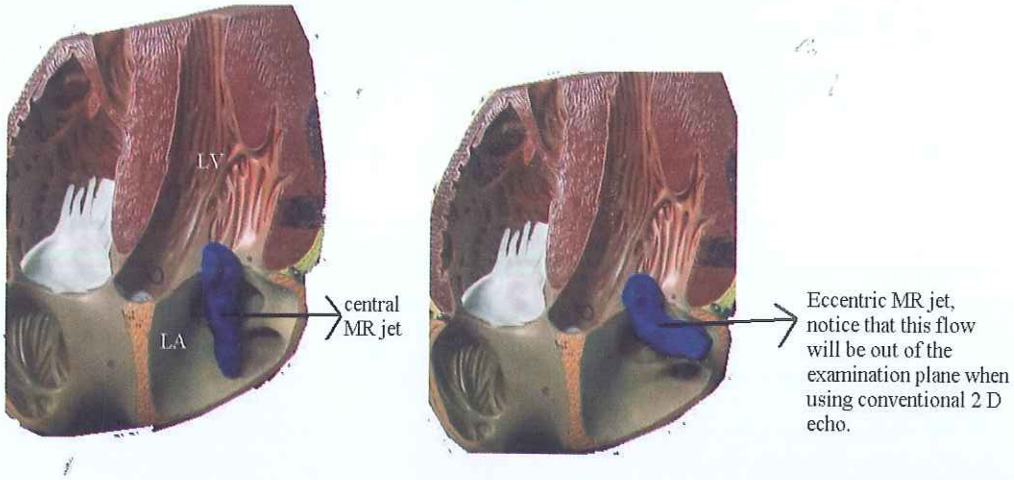
1) Eccentric mitral regurgitation:

In eccentric regurgitation jets, the true amount of the regurgitation jet will be underestimated, see the 3D illustrated picture in the diagram. For example, a four chamber view will only show lateral and medial orientation, with failure to show an anterior and posterior orientation, while an apical 2 chamber view and a PLAX view will show both the anterior and the posterior orientation with failure to clear out the lateral and medial directions. So it is a common practice to upgrade the severity of the regurgitation jet by one degree if it is proved to be eccentric.

In diagram 81, in the apical 4 ch view the MR appears falsely moderate, because we cannot see the posterior amount of the jet (which will be posterior to our 2 D scan beam. i.e. behind the screen), but by imaging the PLAX view we are able to see the anterior and posterior orientation and the true severity of the MR is revealed.

Diagram 80

3 D diagrammatic model for central and eccentric mitral regurgitation(MR) jet.


LV
LA
central MR jet
Eccentric MR jet, notice that this flow will be out of the examination plane when using conventional 2 D echo.

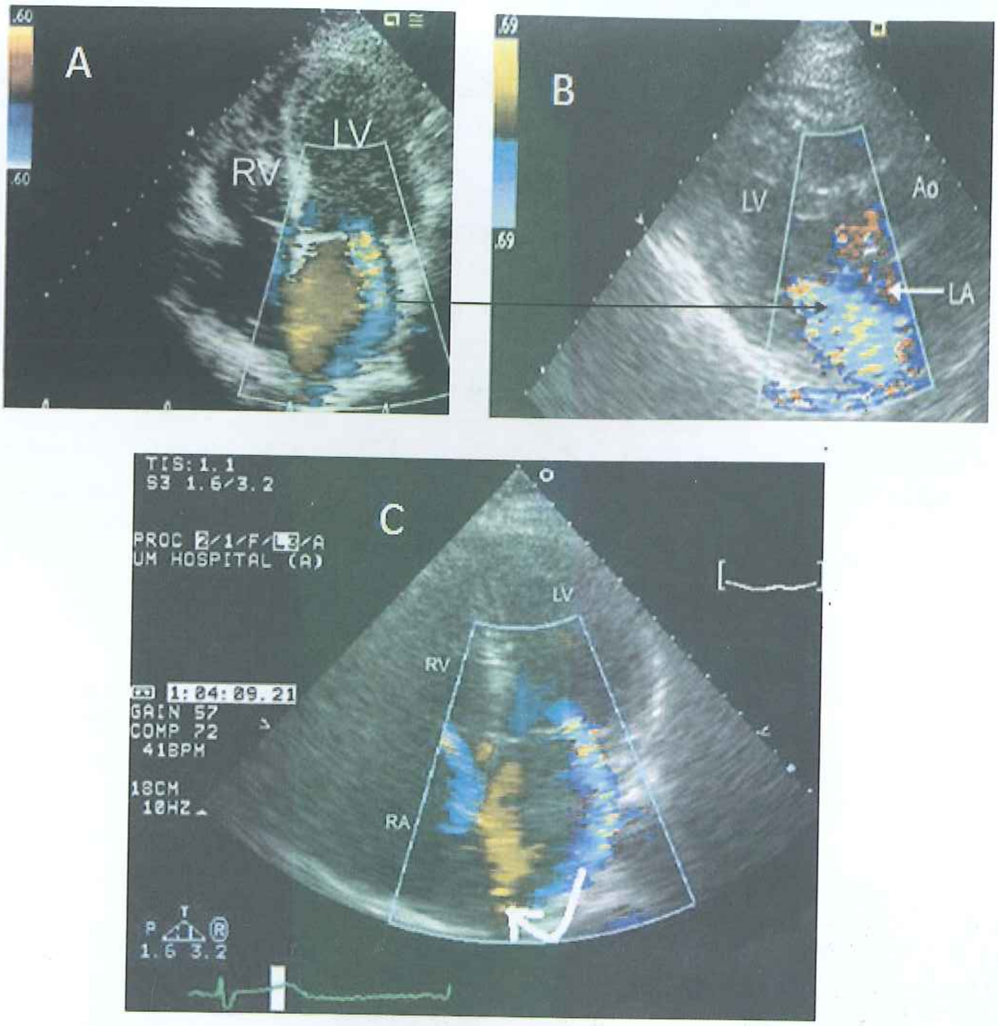


Diagram 81 A : excentric mitral regurgitation is seen in an apical 4 chamber view, B :the actual amount is revealed in the PLASX. See text for more details.

81 C : another case of severe excenteric mitral regurgitation, where the severe jet hits the left atrial wall and, this results that the jet is directed backwards as shown by the arrow " Swirling effect."

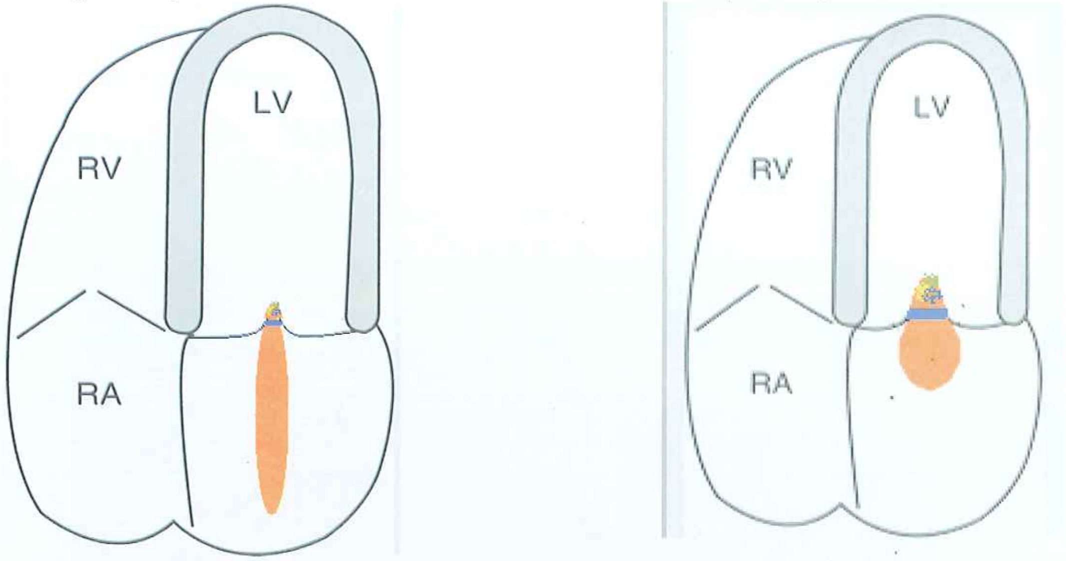
So, it is mandatory to examine the regurgitation jet in multiple views and multiple planes. Multiple plans will be examined by tilting motion in any view in order to scan the left atrium for any high velocity regurgitation jet. (Revise tilting motion from "echocardiography views).

N.B vena contract is difficult to measure in eccentric jets.

2) Poor cooptation of the mitral valve:

Remember from chapter "clinically applied physics" that the color Doppler describes the flow in terms of velocity, so with poor cooptation the regurgitation jet will pass through a relatively wider regurgitate orifice, this will lead to a relative reduction in its velocity, with subsequent decrease in the amount of the mosaic jet and under estimation of the severity of the regurgitation. See diagram 82.

N.B vena contracta is a good measure in this situation.


A : good cooptation of the M.V

B : poor cooptation of the M.V

Diagram 82 : Diagramtic illustration for the effect of poor coopitation over the amont of the regurgitaent jet surfec area, B : poor copitation of the mitral valve leads to a small regurgitation jet surface area ***(BUT WIDE VENA CONTRACTA " the blue line")*** so it may apear as if it is a mild to moderate case of MR while actually it is a severe case of MR.

3) Poor ejection fraction and patients with low Blood pressure:

In this case the driving force of the regurgitant jet will be relatively low. This will lead to a relative reduction of the velocity of the regurgitatant jet, with a reduction in the amount of the mosaic jet and subsequent false underestimation of the regurgitation. (I.e. the same effect of the poor cooptation of the M.V)

Vena contracta is valid in this situation.

4) Patients with high blood pressure:

This is the opposite of the previous condition, the driving force of the regurgitation jet will typically increase leading to an increase in the velocity of the regurgitant jet and overestimation of the mosaic pattern.

5) Mitral regurgitation in a case of mitral valve prolapse:

The color Doppler falsely overestimates the severity of the regurgitation.

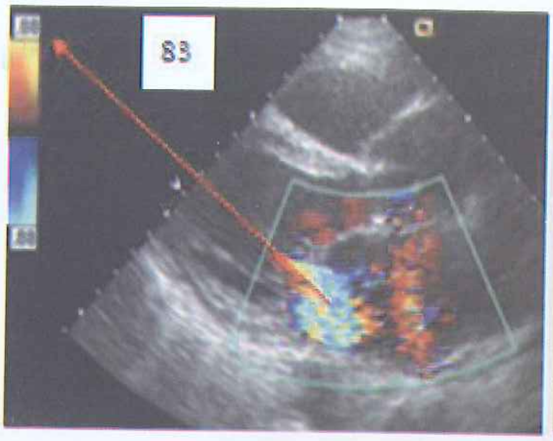
MR regurgitation due to MVP is only confined to the later 50 % of the systole.

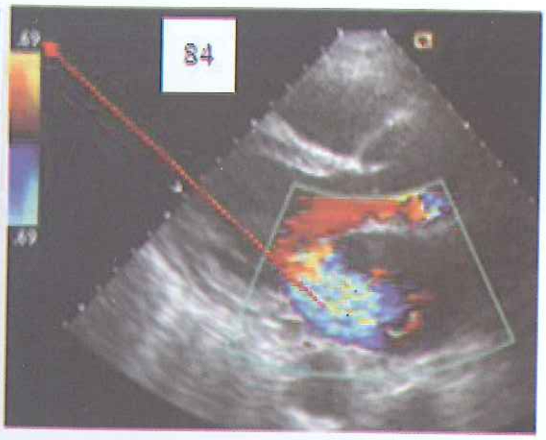
Moderate cases of mitral regurgitation may not cause left atrial dilatation.

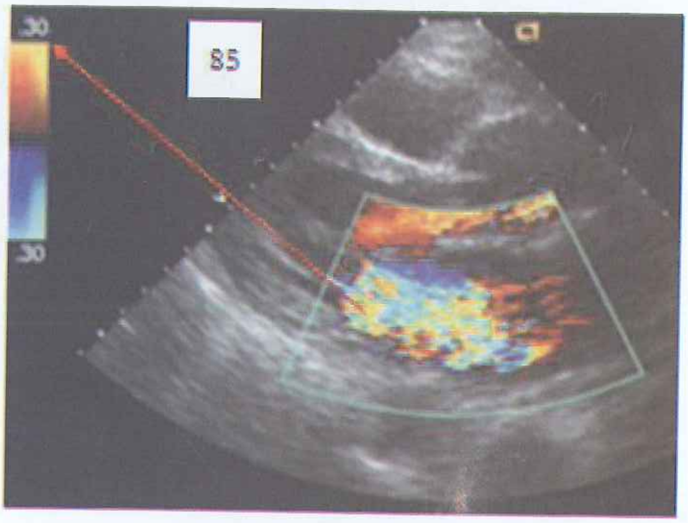
II. Pitfalls related to sonographer's technique:

Nyquist limit (color PRF) :

Notice the increasing jet surface area by the color Doppler in the same patient with decreasing Nyquist limit (See chapter 2). See diagrams below. .







Gain settings: by increasing the gain settings of the color Doppler, the jet surface area will increase, leading to an overestimation of the jet area. The net effect of increasing gain settings will be very similar to decreasing the Nyquist limit, but gain is post processing, while decreasing the Nyquist limit is post processing. (See before)

The Use of different machines to follow up MR or compare results will lead to different grading of severity. (See before)

Important Conclusions:

Owing to the various pitfalls illustrated above, many findings must be integrated together before judging the severity of the mitral regurgitation, these findings include :

1) Percentage of the color Doppler jet area traced out of the LA. (main technique) see diagrams above.
2) Vena contracta
3) Flow reversal in the pulmonary veins (see later).
4) The length of the regurgitation jet into the LA, one third, two thirds or the whole left atrial length.
5) Density of the regurgitation jet using CW Doppler.
6) Patient's clinical condition.
7) Hemodynamic effect of the regurgitation, namely : LV /LA dilatation and pulmonary hypertension.

Clinical scenario:

In a patient with a MR that comprises only 30 % of the LA area, it simply denotes moderate MR, But, if the LA is hugely enlarged, presence of severe PHT, hugely dilated LV, poor LV function or poor patient's clinical status, a professional sonographer must think of a pitfall that faulty underestimated the percentage of the traced jet area.

In a patient with a border line value (mild to moderate, or moderate to severe), the presence of one of the signs suggesting severity illustrated above (3-7) will encourage the sonographer to report as the higher suspected grade.

N.B :

In our school in the **EMSE,** we advise that a professional sonographer has to be decisive in most of his reports.

Aortic regurgitation :

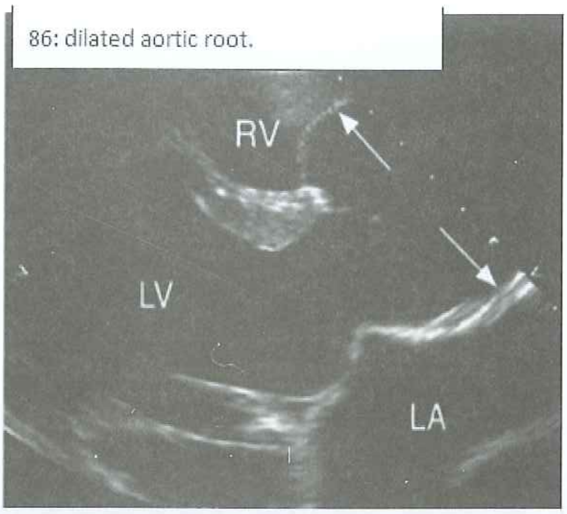
Causes of aortic regurgitation :

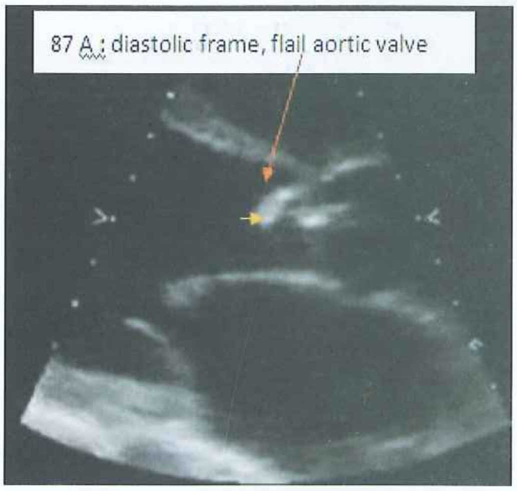
Leaflet abnormalities	Aortic root or ascending aorta abnormalities
Rheumatic fever	Age-related aortic dilation
Infective endocarditis	Annuloaortic ectasia
Trauma	Cystic medial necrosis of the aorta (isolated biscuspid aortic valve or Marfan's syndrome)
Myxomatous degeneration	Systemic hypertension
Congenital aortic regurgitation	Aortitis (syphilis, giant cell arteritis)
Systemic lupus erythematosus	Reiter's syndrome
Rheumatoid arthritis	Ankylosing spondylitis
Ankylosing spondylitis	Behçet's syndrome
Takayasu's arteritis	Psoriatic arthritis
Whipple's disease	Osteogenesis imperfecta
Crohn's disease	Relapsing polychondritis
Drug induced valvulopathy	Ehlers-Danlos syndrome

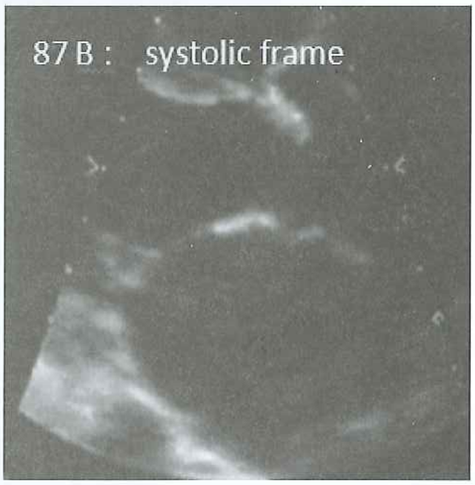
Echocardiography findings :

2D imaging :

The cause of the AR may be evident on the 2 D imaging. For example a bicuspid aortic valve may be seen in the PSAX view (see before). A dilated aortic root should not be missed (diagram 86). A flail aortic valve can also be seen in a PLAX view (diagram 87 A). A vegetation can be seen in any view showing the Aortic valve (diagram 87, small arrow pointing to the vegetation) . Poor cooptation of the aortic valve can be seen in PSAX aortic valve level. (diagram 89). See diagrams 86, 87, 88, 89.


86: dilated aortic root.


87 A : diastolic frame, flail aortic valve


87 B : systolic frame

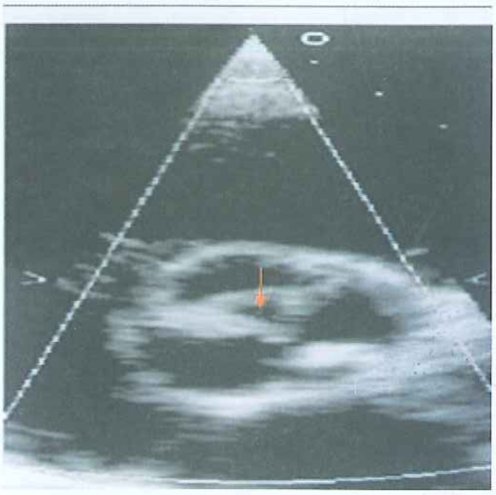


Diagram 88: Zoom in PSAX aortic valve level, showing poor coopitation of the aortic valve shown by the arrow.

The sinus of valsalva can be measured using the 2 D echo as well as the aortic root, sinotubular junction, and the ascending aorta and should be reported. See diagram 90.

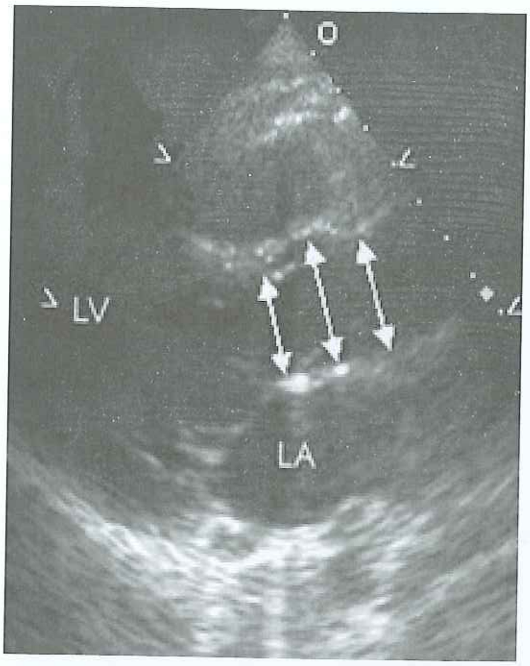


Diagram 90 : Exact sites to measure (from left of the screen to the right) the: 1) aortic root, 2) sinus of valsalva and 3) sinitubular junction

M.mode:

The closure line may show poor cooptation.

An eccentric line of closure should raise suspicion for a bicuspid aortic valve. (See before)

Color M.mode also confirms the diagnosis with very good specificity but less sensitivity .

See diagram 91 A & B.

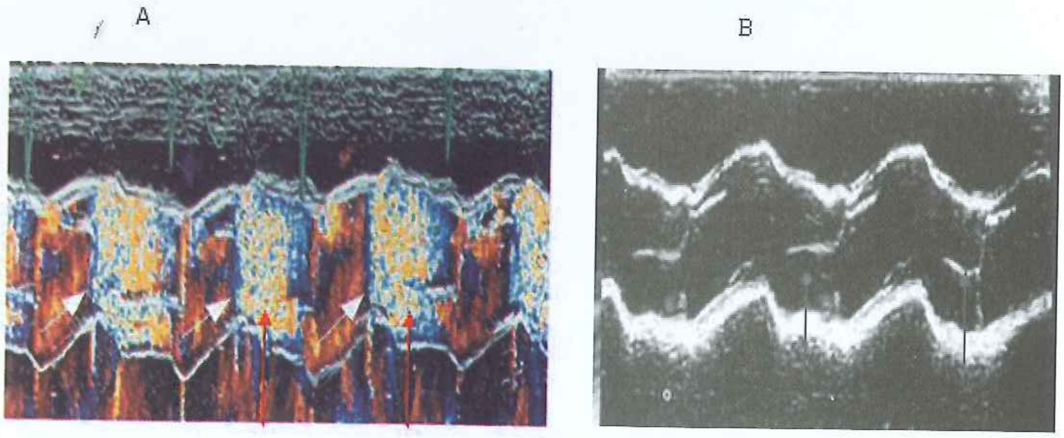


Diagram 91 : A : severe aortic regurgitation, the arrows are pointing to the closure lines, at the closure line, normally, there should be no color trace, the color trace on the closure line denotes aortic regurgitation. B : normal closure line of the aortic valve.

Fluttering of the anterior leaflet of the mitral valve:

This occurs mainly with severe aortic regurgitation or posteriorly directed jets that will hit the mitral valve during the diastole (i.e. during the opening of the mitral valve), this will lead to partial valve closure but the forward flow is still ongoing !. Here the anterior mitral valve leaflet is being subjected to two flows, the normal flow is trying to open it, and the AR jet is trying to close it. This will finally lead to fluttering of the anterior leaflet of the MV. This finding is easily seen by the M.mode study as a characteristic "zigzag shaped E A waves and "zigzag shaped" EF slope of the mitral valve.

Doppler study:

Color Doppler is the main technique to quantify for a case of aortic regurgitation.

Using the color Doppler , the high velocity aortic regurgitation jet can be seen in any view opening the aortic valve, for example: PLAX, apical 5 chamber and apical 3 chamber views.

See diagrams 92, 93, 94 , 95, 96, 97, 98, 99, 100. The regurgitation jets are pointed to by arrows.

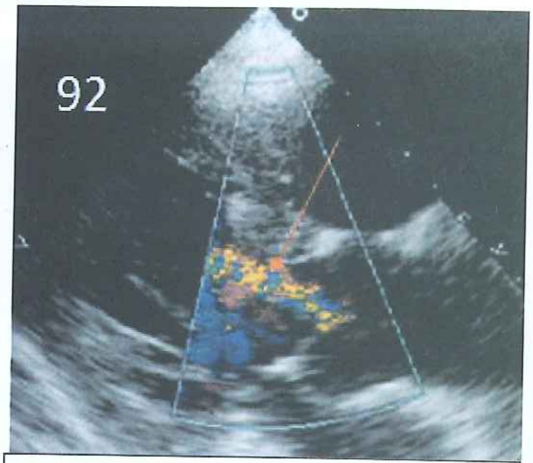


Diagram 92 : Mild aotrtic regurgitation, the regurgitation. The regurgitation jet occupies 20 % of the LVOT.

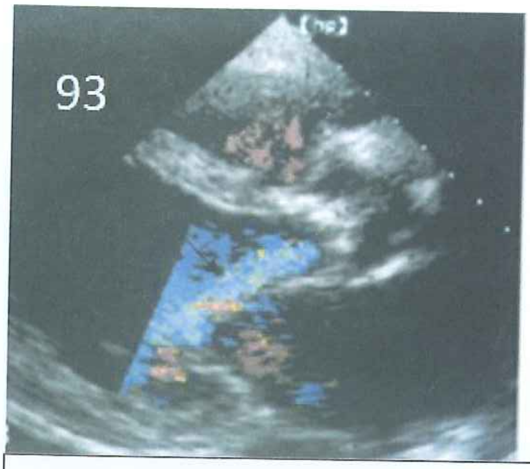


Diagram 93 : moderate aortic regurgitation, the regurgitation jet occupies 50 % of the LVOT. (Notice that the different jet direction will be encoded in different colors.)

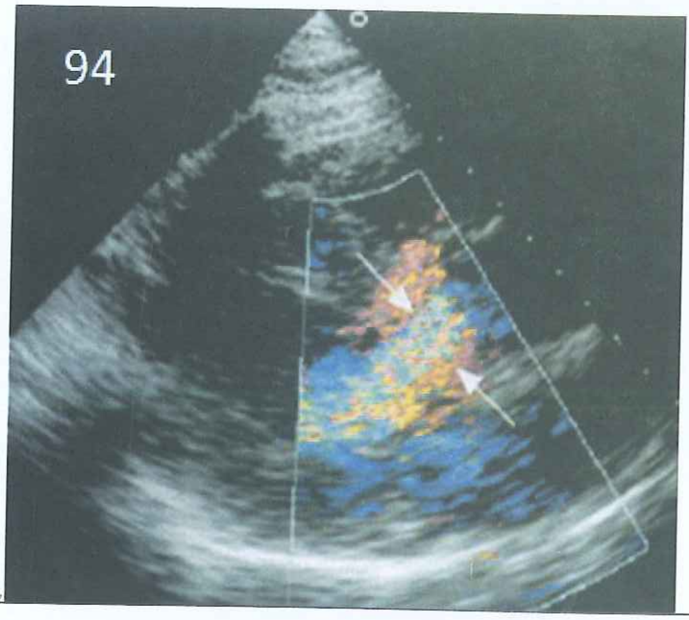


Diagram 94 : severe aotrtic regurgitation, the regurgitation.

The regurgitation jet occupies 90 % of the LVOT. The jet is measured as shown in this digram.

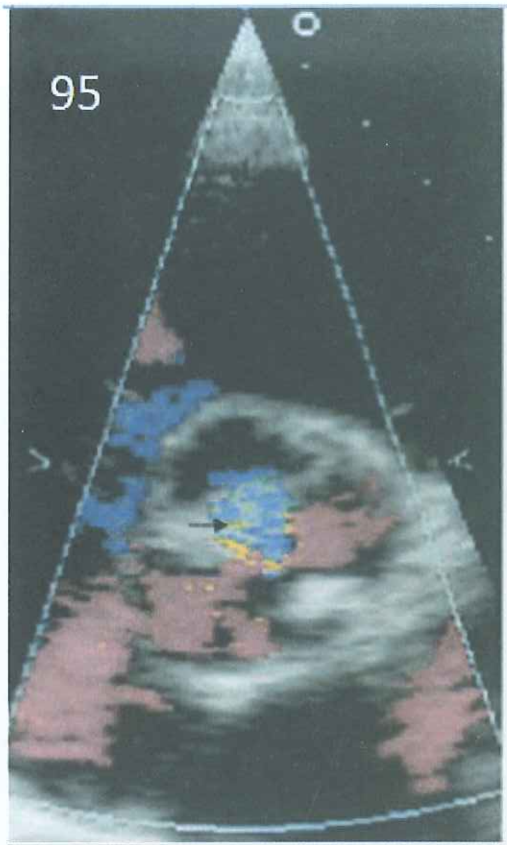


Diagram 95 : Aortic regurgitation in PSAX view aortic valve level , better to be judged in a PLAX or apical 4 ch view

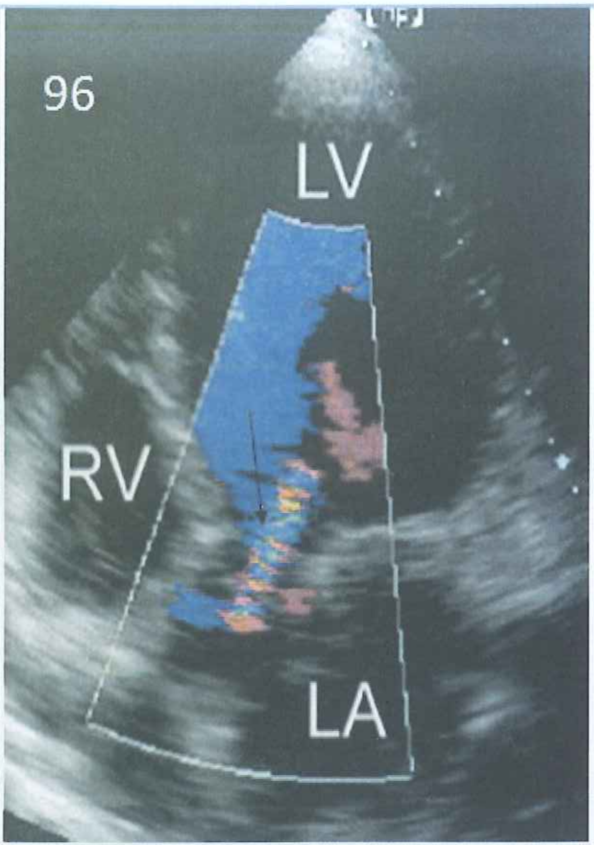


Diagram 96 :mild Aortic regurgitation in apical 5 chamber view .

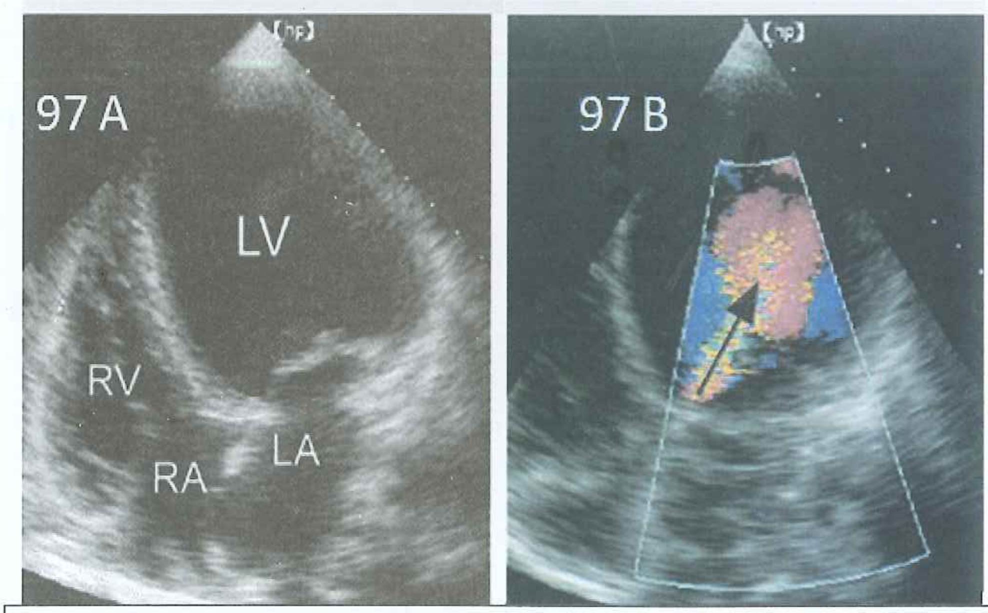


Diagram 97 A &B : posterior directed aortic regurgitation may be clear in an apical 4 ch view too. A : without color Doppler , B: same view with color Doppler.

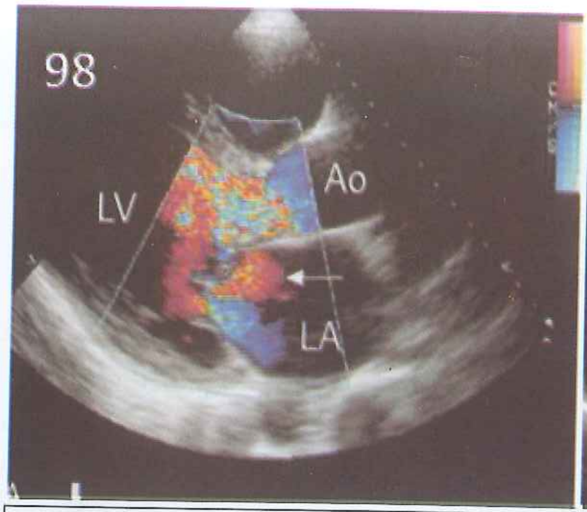


Diagram 98 : uncommonly, in severe cases of aortic regurgitation, the regurgitation jet hits the LV wall and reflects back into the LA. Causing what's called : Diastolic mitral regurgitation

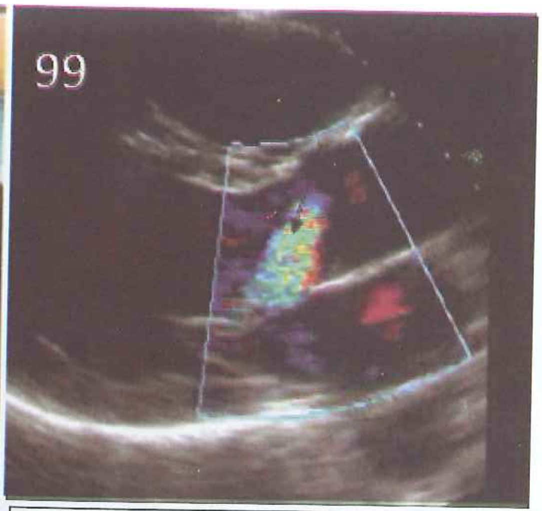


Diagram 99: severe case of aortic regurgitation, the regurgitation jet hits the anterior leaflet of the mitral valve, this will cause fluttering in the anterior leaflet of the mitral valve.

Generally in an apical view the color Doppler correlates well with the amount of regurgitated blood. By aligning the color spectrum over the basal part of the LV, the AV and most of the LV, the red mosaic regurgitation jet can be elicited. Unlike the mitral regurgitation, aortic regurgitation correlates better with the percentage of the width of the flow of the LVOT

See previous diagrams for grading the severity.

The regurgitation jet is a high velocity jet, owing to the Aorta / LV pressure difference during the diastole.

The **EMSE** describes the high velocity jet as being mostly ellipsoid in shape with well delineated borders, and lasts for the diastolic time, i.e. not flashy. A flashy non ellipsoid jet will be mostly a ghost artifact.

N.B the color of aortic regurgitation will vary according to the view and according to the angulation. See diagram.

Vena contracta:

Vena contracta is the narrowest part of the regurgitation jet just downstream the valve. Vena contracta is shown in diagram 100.

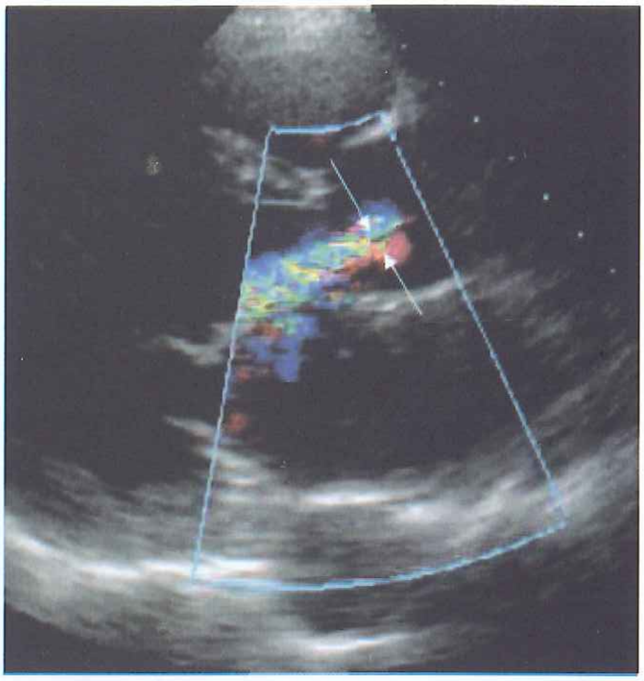


Diagram 100 : PLAX view showing aortic regurgitation. The arrows are pointing to the vena contracta.

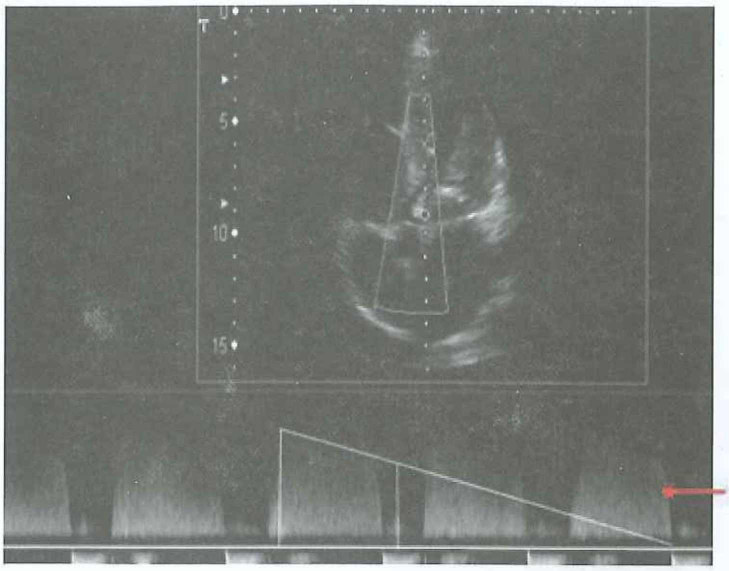
N.B : Grading of severity of aortic regurgitation by vena contracta is done as in mitral regurgitation.

CW Doppler :

In cases of aortic regurgitation, a high velocity +ve deflection signal can be seen by aligning the curser line over the aortic valve in any apical view with the aorta opened (diagram 101). However a well delineated jet with sharp borders is difficult to get in almost 60 % of the patients.

The density of the signal subjectively correlates with the severity of the regurgitation.

Pressure half time can be used to judge the severity of the regurgitation. In this case, a mild aortic regurgitation will pass backward from relatively narrow regurgitate orifice area so the pressure half time will be relatively long, latter on in cases of severe aortic regurgitation, the orifice will relatively dilate causing the pressure half time to shorten. So if Pressure half time is >500 msec this correlates with mild aortic regurgitation, while if the pressure half time is <250 msec it correlates with sever aortic regurgitation.



Aortic regurgitation signal

Diagram 101: Pressure half time is used to estimate the severity of aortic regurgitation.

N.B : Many pitfalls complicate this measure, so it is not included in the recent echocardiography guide lines.

Acute aortic regurgitation will cause marked shortening in the pressure half time.

Very severe aortic regurgitation will cause marked prolongation in the pressure half time.

Any abnormalities in the patient's blood pressure will affect the pressure half time.

PW Doppler is of little value in AR, and can only be used to localize the origin of the high velocity aortic regurgitation jet in some situations.

Pitfalls in assessment of aortic regurgitation using color Doppler :

Nearly the same general pitfalls of color Doppler (including pitfalls discussed in MR) are included as pitfalls in assessment of aortic regurgitation.

A) **Pitfalls due to poor echocardiographer technique :**

1) Atypical views: any atypical view will lead to under estimation of the severity of the regurgitation lesion, in mild cases of AR, an atypical view may lead to overlooking of the regurgitation jet and miss diagnosis.
2) Low Nyquist limit will over estimate the regurgitation jet, while high Nyquist limit may lead to under estimation.
3) High color gain settings and low color gain settings will increase or decrease the regurgitation jet, respectively.

B) **Pitfalls related to patient's clinical condition:**

1) Medical conditions that affect the heamodynamics will affect the size of the jet, for example: in hypotension the driving pressure of the aortic regurgitation will be lower than that of normotensive patient , so the velocity will be relatively lower than a normotensive patient, this will decrease the amount of the color mosaic in the LVOT which may lead to false underestimation of the jet.
2) The opposite occurs in hypertension, where the driving pressure in the aorta will be abnormally high, leading to a higher velocity jet than in normotensive patient, this relatively higher velocity jet will increase the amount of the color Doppler and may lead to a false over estimation.

N.B : during assessment of aortic regurgitation it is mandatory to record the blood pressure before the examination.

3) Poor cooptation of the aortic valve:

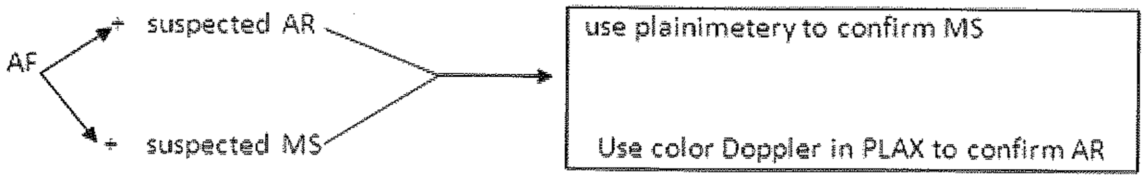
This will lead to a larger regurgitation orifice area "ROA"(however there is a measurement called effective regurgitation orifice area EROA, but the details of the EROA is out of the scope of this book.),
This larger ROA will lead to slight reduction in the velocity of the regurgitation jet, which may lead to false underestimation.

Valvular lesions + Atrial fibrillation (AF) :

The main caveat is confusing the M.S signal as A.R while aligning the CW Doppler over the aortic valve.
Remember that in cases of AF, the "A" wave of the mitral flow will be lost, so, the mitral stenosis will look like the decrescendo wave of the AR.
During AF and a real case of mitral stenosis, the CW Doppler aligned over the aortic valve may be contaminated by the MS signal, leading to a false impression for the beginner echocardiographer as if the patient suffers from a high velocity jet across the LVOT and aortic valve (false impression of AR). So in suspected cases of AF and AR, the beginner ehcocardiographer should confirm the presence of AR by color Doppler in the PLAX view (as in diagram 100)

The opposite situation may occur, during AF and a real case of aortic regurgitation, the CW Doppler aligned over the mitral valve may be contaminated by the aortic regurgitation signal, leading to an impression for the beginner echocardiographer as if the patient suffers from a high velocity jet across the mitral valve. So in suspected cases of AF and M.S, the beginner ehcocardiographer should confirm the presence MS by planimetry in the PSAX view MV level.

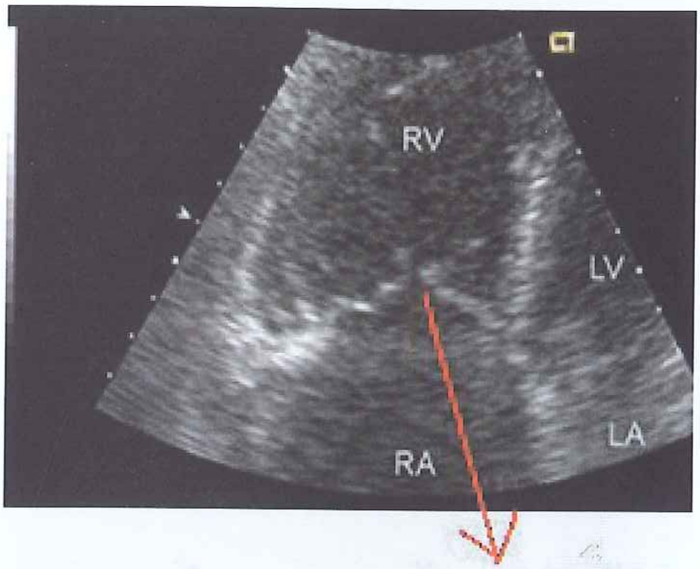
EMSE clinical tip for beginner echocardiographer


AF
+ suspected AR
+ suspected MS
use plainimetery to confirm MS
Use color Doppler in PLAX to confirm AR

Tricuspid regurgitation "TR" and Assessment of the pulmonary artery systolic pressure

2D imaging :

2 D imaging may show some clues to the cause of the TR, an associated dilated RV will cause tethering in the TV and lead to TR, also poor cooptation of the TV may be evident by 2 D imaging. TV prolapse was also documented in some clinical scenarios and lead to TR.



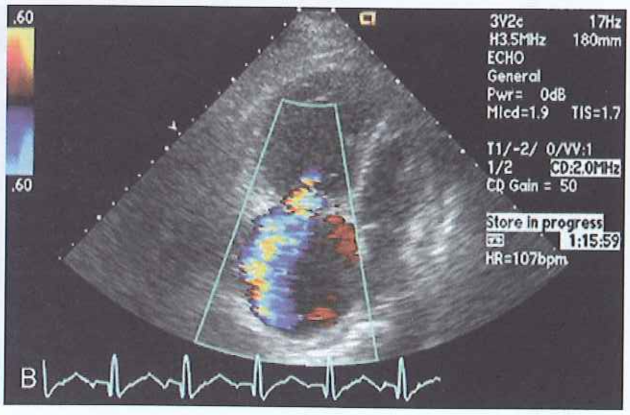
notice the poor cooptation of the TV

M.mode : of little value.

Doppler study :

Exactly the same concepts used to asses MR will be used to asses TR.

Color Doppler will be used to assess the severity of the tricuspid regurgitation using the same numerical values for MR.

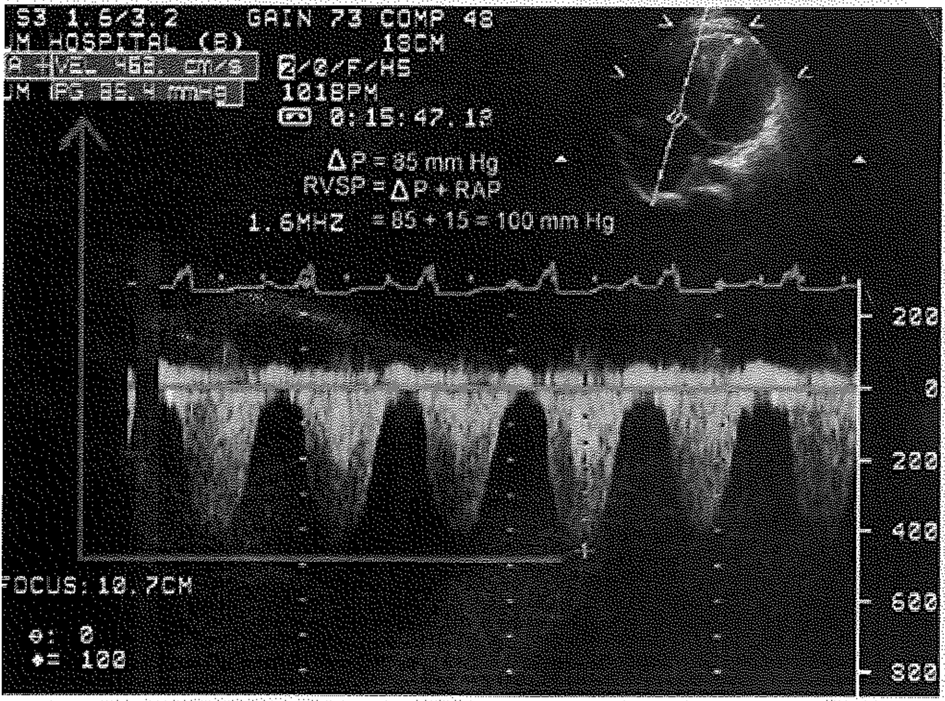


color Doppler showing severe TR.

Assessment of the estimated pulmonary artery systolic pressure (EPASP) "Pulmonary hypertension":

PASP = peak TR PG + 5 mmHg

(It is a common practice for many echocardiography laboratories to fix a certain number for the estimated right atrial pressure "RAP" to be added to the peak TR PG, some echo labs estimates the RA pressure as 5 mmHg, 10 mmHg or up to 15 mmHg. However, the accurate estimation of the RAP should be based on the IVC size)



CW Doppler aligned over the TV showing –ve Doppler signal of TR.

The PPG measured by the sonographer equals 85.4 mmHg {4(4.62^2)}, the EPASP will equal to 5 mmHg + 85.4 mmHg. So the EPASP = 90.4 mmHg

Explanation :

During the systole the pulmonary valve (PV) is open; this will lead to equalization of pressure between the pulmonary artery (PA) and the right ventricle (RV); assuming no pulmonary stenosis.

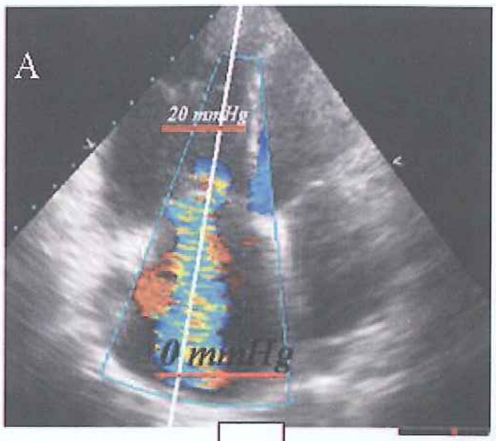
So by measuring the RV pressure we are actually measuring the PA pressure.

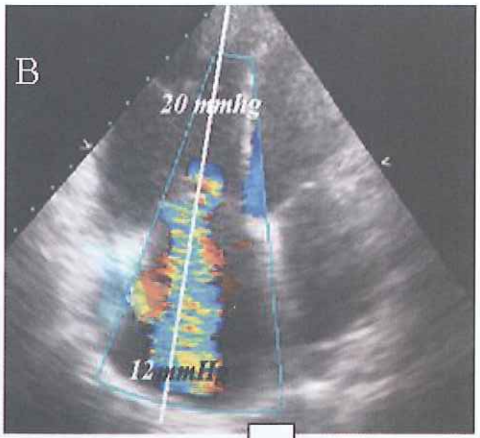
In the presence of TR, the tricuspid regurgitation jet will flow into the right atrium with pressure that equals the right ventricular pressure (which in turn is equal to the PA pressure).

Assuming that the RA pressure is zero, then the pressure gradient (pressure difference) measured between the RV and the RA will be equal to the RV pressure.

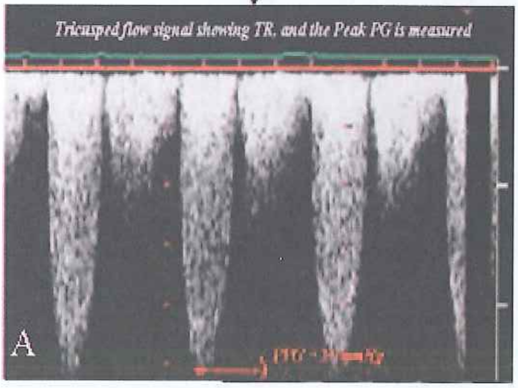
Illustrative example:

A: An assumption is made that the RA pressure is zero mmHg | **B:** An assumption is made that the RA pressure is 12 mmHg

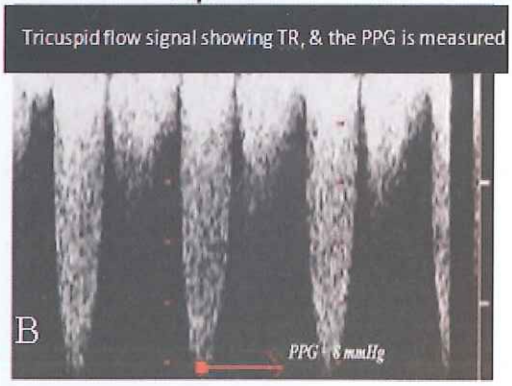




A: An assumption is made that the RA pressure is zero mmHg | B: An assumption is made that the RA pressure is 12 mmhg


Tricusped flow signal showing TR, and the Peak PG is measured
A
PPG = 20 mmHg

PPG= 20 mmHg



PPG = 8 mmHg

A : Assuming that the RA pressure is zero, then the pressure gradient between the RV and RA equals the RV pressure. PG = RV-RA *→20 = RV- 0 *→ RV = 20 mmHg. (The PPG of the TR= RV pressure)

But what if the RA pressure is not zero?!, in this case the PG measured by the CW Doppler between the RV and the RA will not equal to the RV pressure. Rather, it will be underestimated by a value equivalent to the RA pressure.

B : Assuming that the RA pressure is 12, then the pressure gradient between the RV and RA will not equal to the RV pressure. PG = RV-RA *→20 = RV- 12 *→ RV = 8 mmHg (RV= PG + RAP). In this example, if the echocardiographer faulty assumed that the RAP is zero then the calculated RV pressure will be underestimated. So what should the echocardiographer do to obtain an accurate RV pressure? The echocardiographer has to add the RA estimated pressure "12 mmHg in this example" to the measured tricuspid regurgitation PPG. So the estimated RV pressure will be equal to 12 mm Hg + 8 mmHg

Estimated RV pressure = 20 mmHg

Therefore, the estimated right ventricular pressure = TR PPG + estimated RA pressure.

In normal situations the RA pressure is assumed to be 5 mmHg.

For more accurate results the RA pressure can be estimated from the IVC dimensions and being collapsible or not.

Normally the IVC should not exceed 1.6 cm in diameter as measured in a bicaval view.

Normal dimension collapsible IVC correlates with 5 mmHg RA pressure.

Dilated collapsible IVC correlates with 10- 15 mmHg RA pressure.

Dilated non collapsible IVC correlates to 15- 20 mmHg RA pressure

Other methods to assess the pulmonary artery pressure less used in clinical practice or used in special situations:

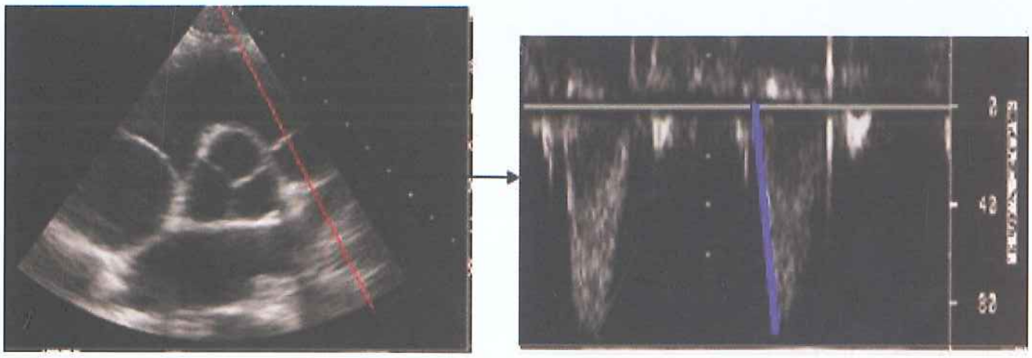
1) **Assessment of the pulmonary vascular resistance "PVR" :**

PVR= {tricuspid regurgitation velocity / VTI of the RVOT} X 10 + 0.16

The PVR is low in cases of increased pulmonary artery pressure due to increased pulmonary blood flow, and this measurement will be high in cases of increased pulmonary artery pressure due to elevated pulmonary vascular resistance.

A low PVR is defined as being less than 0.2.

2) Systolic Pulmonary arterial flow acceleration time: this measure is performed exactly like the pressure half time but the slope is adjusted from the base line down to the apex of the pulmonary artery systolic flow.



Pulmonary artery flow, notice the blue slope used to measure the pulmonary acceleration time.

In pulmonary hypertension the pulmonary acceleration time will typically decrease. The normal pulmonary artery acceleration time is more than 140 msec. (100 msec in some studies)

3) **Mean Pulmonary artery pressure** = {PASP + 2 PADP} /3
   PASP : pulmonary artery systolic pressure (assessed from the peak TR P.G + 5 mmHg)
   PADP : pulmonary artery diastolic pressure (assessed from the peak PR P.G + 5 mmHg)

Pitfalls in the assessment of the EPASP:

- In the presence of pulmonary stenosis the RV pressure will not be equal to the PA pressure during the systole, so the RV pressure measurement does not reflect the PA pressure and the PA pressure cannot be assessed.
- In the presence of poor cooptation of the TV, the velocity of the regurgitation jet will decrease. This will underestimate the calculated TR PPG.
- False estimation of the RA pressure will either over estimate or under estimate the calculated EPASP.

What is normal and what is abnormal :

The definition of pulmonary hypertension is postulated only by the invasive right heart catheterization, as mean pulmonary artery pressure less than 25 mmHg.

In echocardiography far more results were seen in otherwise healthy individuals. The European society of echocardiography postulated that pulmonary artery pressure may be as high as 35 mmHg (adding on the RA pressure).

However a moderate case of mitral stenosis will increase the pulmonary artery pressure to a range between 30 to 50 mmHg.

EMSE clinical insights derived from the guidelines:

So in the **EMSE**, we recommend that if the EPASP is border line i.e. above 30 mmHg, the other echocardiographic criteria and the clinical condition will direct the decision either towards right heart catheterization or towards the "no need to worry" decision.

With border line EPASP and any other positive echocardiographic criteria in terms of: 1)more than mild TR, 2)dilated RA or RV, right heart catheterization is advised if pulmonary hypertension is clinically suspected.

EMSE **classification of pulmonary hypertension:**

Cardiac causes :

Any cardiac abnormality can lead to pulmonary hypertension:

1) Valvular lesions especially mitral valve stenotic and regurgitation lesions.
2) Left to right shunt lesions: ASD, VSD, PDA, PAPVR.
3) Systolic and diastolic cardiac dysfunction

Extra cardiac causes

Chest conditions, ex. COPD

Chronic thrombo-embolic pulmonary hypertension

Primary pulmonary hypertension

Pulmonary veno-occlusive disease

Pulmonary hypertension with unclear/ multi factorial mechanism. Ex, hematological disorders and glycogen storage disease.

EMSE insights on the regurgitation lesions.

For the left sided intra cardiac valves, for every severe regurgitation there must be a "flow reversal":

In severe mitral regurgitation the reversal of flow will be in the pulmonary veins.:

Normally in an Ap 4ch view, placing the sample volume of the PW Doppler in the pulmonary vein, the pulmonary vein will show two positive waves, S and D waves, which represent the normal forward filling from the pulmonary veins to the Left atrium (see diagram 123). Severe mitral regurgitation will lead to flow reversal, i.e. from the LA to the pulmonary vein, this will be represented as a reversed S wave.

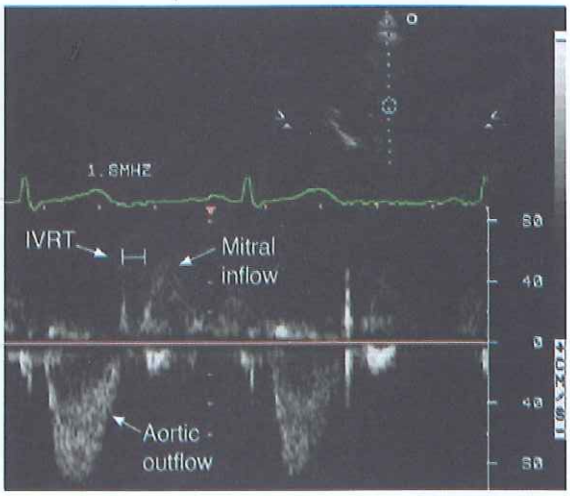
In severe aortic regurgitation (AR) the reversal of flow will be in the descending aorta:

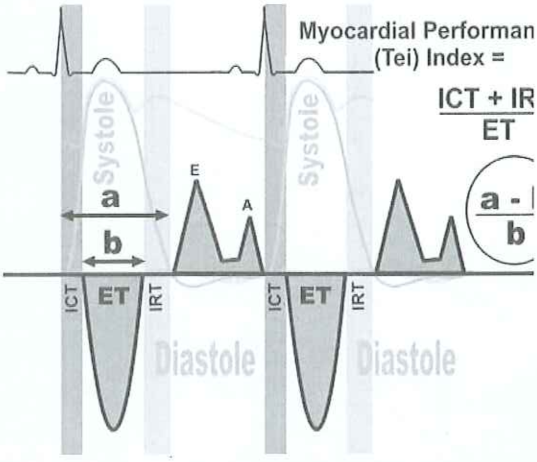
In a SSLA view, the flow of the descending aorta can be detected by CW Doppler, typically this flow will be represented as a negative deflection wave (see diagram 37 B)

In mild cases of AR, only the blood just distal to the aortic valve regurgitates into the LV. With increasing AR severity, the blood more distal to the aortic valve will regurgitates into the LV. With truly severe aortic regurgitation the blood far distal to the aortic valve will regurgitate into the LV, i.e. the blood in the descending aorta will regurgitate backwards into the LV. This will be represented as a low velocity positive deflection signal just after the normal negative descending aorta flow deflection.

(N.B tricuspid regurgitation may cause reversal of flow in to the hepatic veins, but it is of low clinical significance)

Importance of the identification of the isovolumetric contraction time and the isovolumetric relaxation time:





This signal can be elicited by placing the Doppler in-between the LVOT and the mitral valve.

ICT : iso-volumetric contraction time.

IRT: iso volumetric relaxation time.

N.B :

The actual mitral flow does not occur in the IRT, but mitral regurgitation will start from the ICT, this is because the fact that the LV pressure is enormously higher than the LA pressure so it can actually push the blood into the LA in the ICT, but the mitral valve cannot open except in the auxotonic phase i.e. actual diastole, and the mitral valve never opens in the IRT.

The same concept applies to AR. The aortic diastolic pressure is enormously higher than the LV pressure during the diastole, this means that the aorta can push the blood back to the LV in the IRT, but aortic flow cannot occur in the ICT because the aortic end diastolic pressure is 80 mmHg, so, the LV cannot push the blood into the aorta in the ICT, but it can only do this during the actual systole.

As a conclusion :

Mitral regurgitation will start in the ICT. I.e. immediately after the end of "A" wave.

Aortic regurgitation (AR) will start in the IRT. i.e. immediately after the end of the normal aortic signal.

This can be used to aid to differentiate between the MR signal and the AS signal. The AS signal can never occur in the ICT but the MR will occur in the ICT.

Also this can aid to differentiate between MS signal and AR signals (in cases of atrial fibrillation)

MS signal can never occur in the IVR time, but AR will occur in the IVR time.

EMSE summary: any regurgitation signal occurs **immediately** after the end of its normal flow signal.

Pulmonary regurgitation "PR":

2 D imaging :

The cause of pulmonary regurgitation may be evident by 2 D echo, for example a dilated RV and associated pulmonary hypertension may be firstly noticed. These pathological conditions will cause pulmonary annular dilatation and lead to PR.

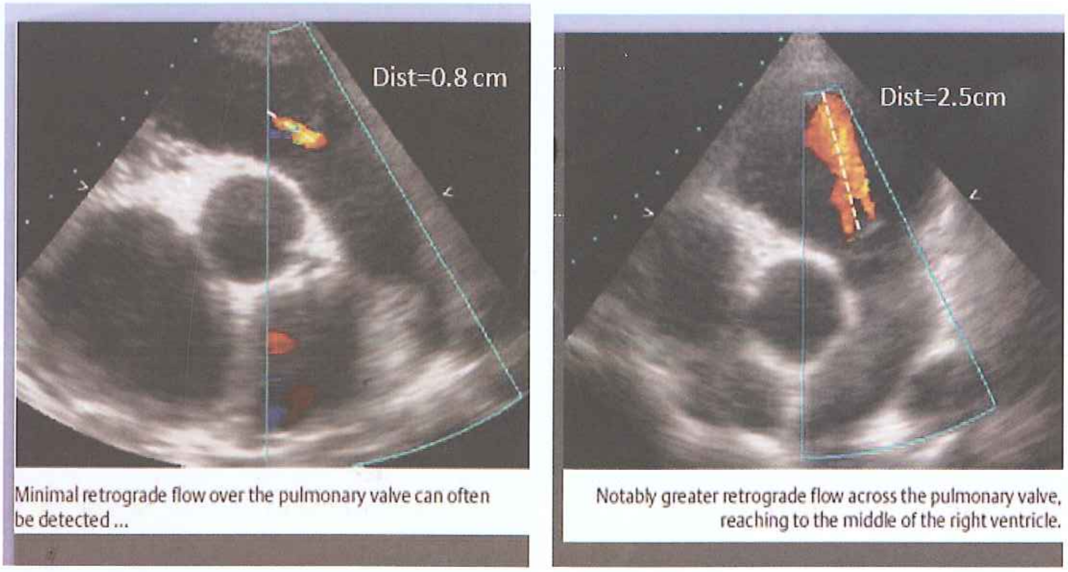
M.mode : of little value.

Doppler study :

Exactly the same concepts used to asses AR will be used to asses PR. The PR signal elicited by the CW Doppler is even the same as the AR signal elicited by the same technique. See diagram 65 for a combined case of PS (-ve signal)& PR (+ve decrescendo signal)

One of the most commonly used parameters is the length of the PR elicited by the color Doppler.

If the length of the PR is less than 1cm report as mild PR, if the length of the PR is more than 1 cm report as severe.



Minimal retrograde flow over the pulmonary valve can often be detected ...

Notably greater retrograde flow across the pulmonary valve, reaching to the middle of the right ventricle.

Pulmonary regurgitation shown in a PSAX great vessels level.

Chapter 8 :Assessment of the left ventricular systolic function and dimensions

Two important aspects must be covered in any echocardiography study :

1) left ventricular functions:

systolic function.

Diastolic function.(see page 180)

2)left ventricular dimensions.

Assesment of the left ventricular systolic function.

2 D echo :

Eye balling:

Eye balling is a subjective method that depends on the sonographer's experience to judge the ejection fraction from a 2D loop video.

With enough experience, it is one of the most important parameters that must be taken into account.

Simpson method " rule of discs"

The concept of this method is that the left ventricle volume is measured by tracing the endocardial border in both the systole and the diastole, then the machine subtracts the systolic volume from the diastolic volume and then dividing the result by the diastolic volume. See diagram 102 .

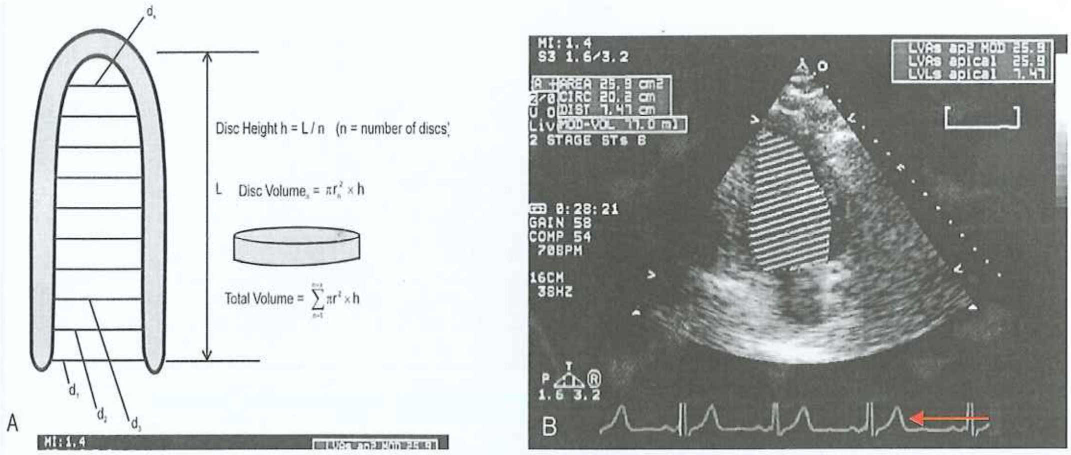


Diagram 102 : Simpson rule : determining the left ventricular volume from the rule of disks . A : The left ventricular cavity has been arbitrarily divided into segments , each of equal height. The height of each cylinder is defined as overall length divided by the number of disks (h = L/n). Individual disk volume is calculated as noted, and the total volume of the ventricle is the sum of the individual disk volumes. Bottom: An actual calculation of the left ventricular volume from an apical two-chamber view.

The main pitfall in this technique is :

1) failure to identify the true endocardial wall, the endocardial wall will only be clear with convenient echo window, sensitive piezoelectric crystals i.e. "new echocardiography machine or a calibrated machine" and experienced sonographer.

2) failure to identify the end systolic frame and the end diastolic frame. This is done much better when the echocardiography study is ECG gated. See the ECG trace pointed to by an arrow in diagram 102.

The main advantage of the Simpson method is that it overcomes most of the pitfalls of the M.mode derived ejection fraction method. See later.

Simpson's method is time consuming, and with the long patients' lists we have in Egypt, it is better to apply it when needed that is, only when the M.mode, eye balling & Doppler methods are dissatisfactory.

Doppler measures to assess the systolic function "stroke volume":

Recall from "Heamodynamics" that by multiplying the VTI (defined in cm) of the LVOT by the cross sectional area of the LVOT (defined in cm2) the resulting number will be the stroke volume in cm3. See diagram 42 and 43.

The normal stroke volume is 70 ml/ beat to 110 ml/ beat.

This measurement is important in many clinical situations, including the diagnosis of heart failure with preserved ejection fraction. HFpEF..

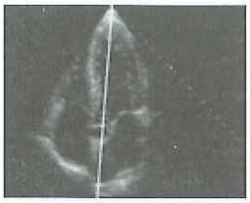
Definition of HFpEF :

1) Normal or fair ejection fraction
2) Symptoms of heart failure, symptoms of pulmonary/ systemic volume over load or low cardiac output symptoms.
3) Impaired ejection velocities, namely the VTI, more precisely the Stroke volume.

HFpEF : the main key for the diagnosis of HFpEF is to measure the Stroke volume (SV).

Important clinical tip : it is mandatory to take extreme care of the fluid chart of a patient with HFpEF, and you must keep this patient euovolemic.

Stroke volume (cm^3) = VTI LVOT by PW (cm) X LVOT area (cm^2)



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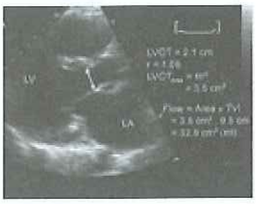
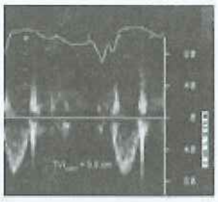




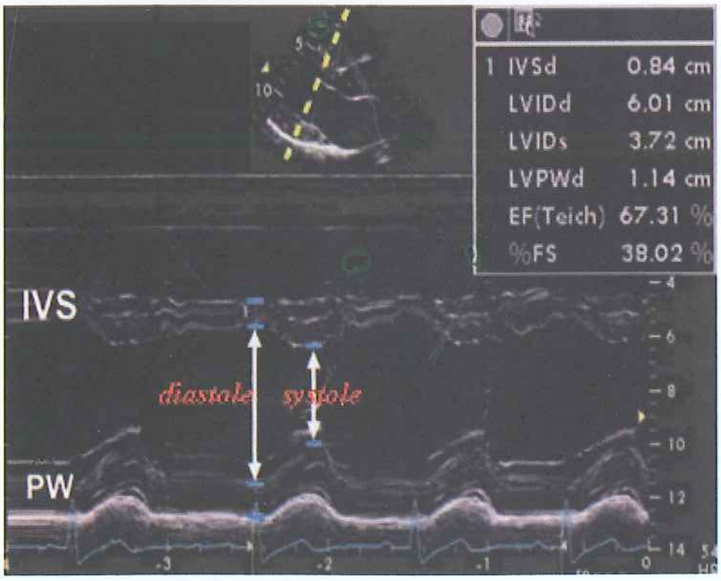
X pi r^2

Diagram 103 : calculating the stroke volume.

M.mode derived ejection fraction :

By measuring the end diastolic diameter and the end systolic diameter the machine will automatically calculate the ejection fraction as $LVEDD^3 - LVESD^3 / LVEDD^3$.

End diastolic and end systolic diameters are defined as the measurement from the true endocardial border of the septum to the true endocardial border of the posterior wall.



1 IVSd 0.84 cm
LVIDd 6.01 cm
LVIDs 3.72 cm
LVPWd 1.14 cm
EF(Teich) 67.31 %
%FS 38.02 %

Diagram 104 : measuring the E.F by M.mode.

Align the M.mode curser perpendicularly over the septum and posterior wall in a PLAX or PSAX. (this is done by selecting the M.mode button in the machine)

Freeze the picture.

Select from the menu the " ejection fraction"

Using the select button, start measuring the different layer of the heart as shown in the diagram 104.

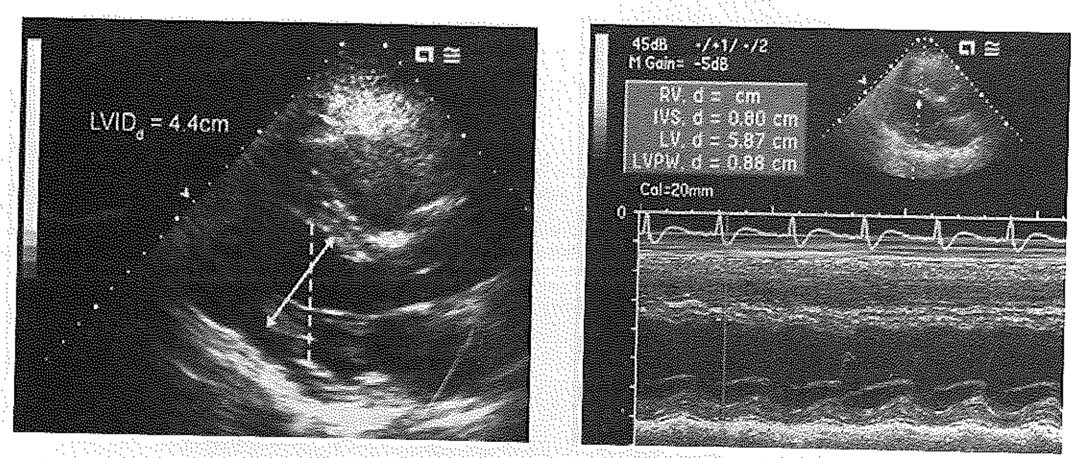
The main pitfalls in this technique :

1)Presence of wall motion abnormality : in this situation the ejection fraction may be underestimated or overestimated, remember from the echo anatomy that the heart is formed out of 16 segment (or 17 segment in other schools). The M.mode measures the E.F from only 2 points from 2 opposing segments. For example, if the patient suffers from septal wall and posterior wall akinesis or hypo kinesis the ejection fraction will be severely underestimated, while if the patients suffers from a lateral wall and anterior wall akinesis or hypo kinesis the posterior wall and the septum may show compensatory hyperkinesis, and the ejection fraction will be overestimated.

2) Slanting cut sections:

* Slanting cuts will lead to an over estimation in the LVEDD, see diagram 105. When this diameter is cubed, the calculated ejection fraction will be reduced.(remember that division affects the calculation more that subtraction).

*Slanting cuts will give a false impression of hypo kinesis, leading to a reduction in the E.F and a false diagnosis of abnormal wall motion abnormalities (Always correlate the impression of abnormal wall motion to the 2D imaging)



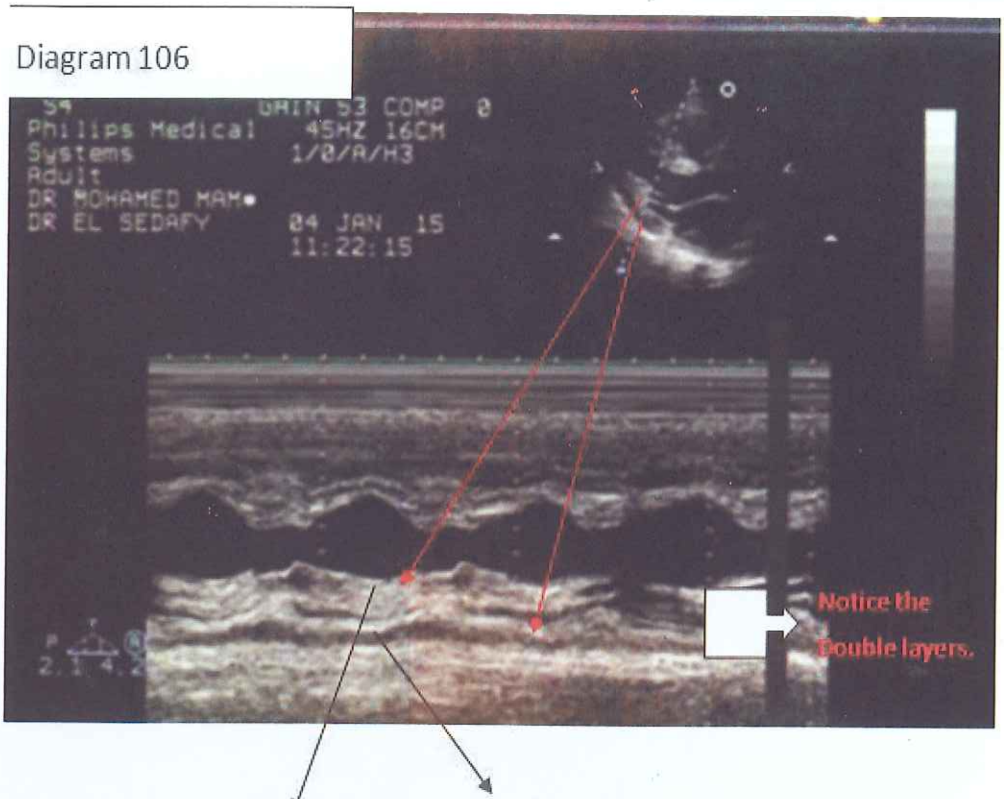
Diagrakm 105 : Santing cut sections will over estimate the LVEDD. (the correct measure is illustrated by the solid line)

To over come this pitfall, ensure that the M.mode curser is aligned perpendicularly over the septum and the posterior wall. This is accomplished by imaging a typical view. i.e. ensure that the PLAX and the PSAX are typical views. (see echocardiographic views)

3) Failure to identify the true endocardial border of the posterior wall:

Normally the mitral valve has a papillary muscle attached to the posterior wall. This papillary muscle will show contractions and relaxations equivalent to the posterior wall systole and diastole complicating the identification of the posterior wall (but remember that the excursion of the posterior wall endocardial border is better than the excursion of the papillary muscle) . See diagram 106, a papillary muscle is encountered by the curser before the actual layer of the posterior wall. The papillary muscle is denoted by an arrow.

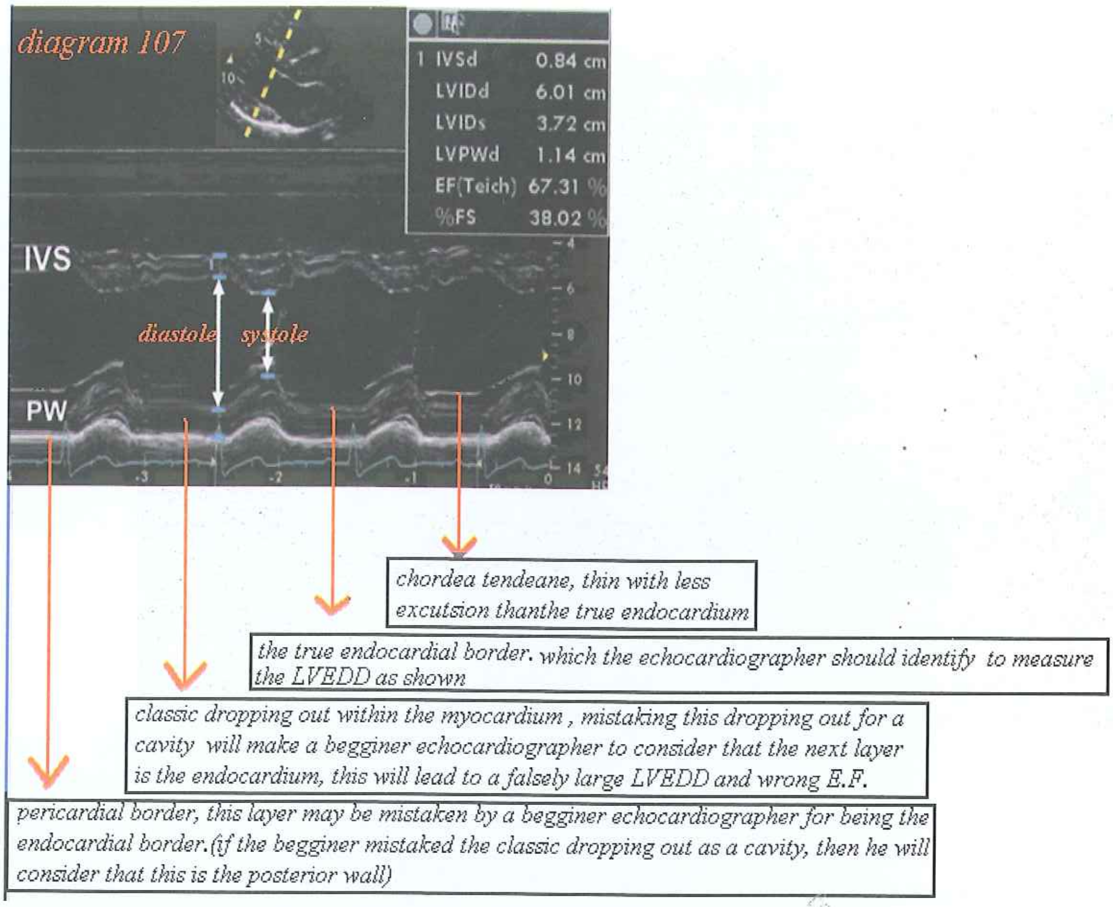
Diagram 106



The endocardial border of the papillary muscle, mistaking this border as the endocardial border of the posterior wall will lead to wrong measurement of the LVEDD, and wrong E.F.

The true endocardial border of the posterior wall. Notice the it is the most white most continuous layer with best excursion.

Also the classic dropping outs within the posterior wall may raise questions about the true border which the sonographer should identify as the true endocardial border. See diagram 107.



chordea tendeane, thin with less excutsion thanthe true endocardium

the true endocardial border. which the echocardiographer should identify to measure the LVEDD as shown

classic dropping out within the myocardium , mistaking this dropping out for a cavity will make a begginer echocardiographer to consider that the next layer is the endocardium, this will lead to a falsely large LVEDD and wrong E.F.

pericardial border, this layer may be mistaken by a begginer echocardiographer for being the endocardial border.(if the begginer mistaked the classic dropping out as a cavity, then he will consider that this is the posterior wall)

Diagram 107 : showing the classic dropping out within the cardiac walls and different cardiac layers that may confuse a beginner echocardiographer.

The **EMSE** tips to overcome the above difficulties:

1) Correlation between the 2D imaging and the M.mode borders is a must. See diagram.
2) The posterior wall endocardial border is defined as the whitest most continuous line **with best excursion**. See diagram.
3) If you are a beginner sonographer:
   * It is better to start your examination with high gain settings, and then gradually decrease your gain settings until the LV cavity becomes black while you are keeping notice of the posterior wall layer not to drop out.
   *Always indentify the pericardium as the outer most layer of the heart, then try to identify the myocardium then the endocardium.

N.B :

*Before making a decision about identification of the posterior wall the sonographer must ask himself a very important question, is this a hypo echoic region within the wall (i.e. classic dropping out) or is it a true cavity?

*Identification of the posterior wall endocardial border is much easier in these two situations :

-when the sonographer optimizes the view

-In patients with normal BMI/ BSA.

EPSS : E point septal separation as an indirect estimation of the LV systolic function.

In normal myocardial systolic function the mitral valve opens with good excursion so that the tips of the mitral valve touches the inter ventricular septum ,as shown diagram. In cases of impaired systolic function and due to abnormal inracardiac heamodynamics the mitral valve excursion is not good, leaving a distance between the tips of the mitral valve and the IVS. So normally the "E " point of the mitral valve should be separated from the septum (IVS) by minimum millimeters, so the normal EPSS is a small value typically < 8mm, and this number increases in impaired systolic function.

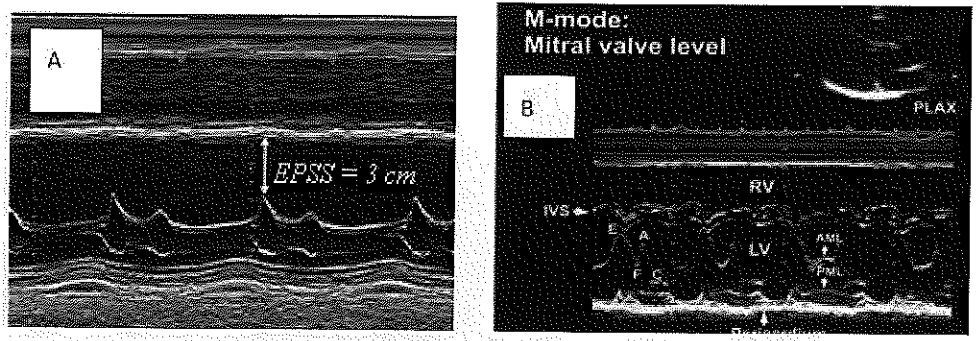


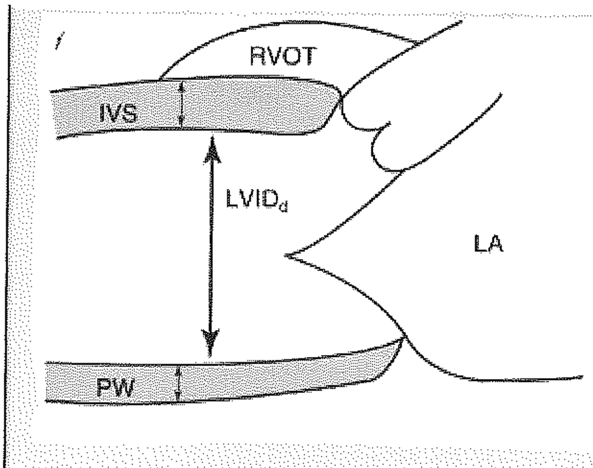
Diagram 108 : A : abnormally large EPSS in a case of impaired E.F, B: normally small EPSS in a normal subject.

Other less commonly used techniques in assessment of systolic function :

Less important Echocardiographic criteria derived during the assessment of the systolic function :

The importance of any echocardiographic measure is consistent with how crucial that measure will affect the clinical scenario, for example, valvular assessment is one of the most critical information upon which a huge decision will be made either to intervene or not. Other echocardiographic data upon which there is a little change in the clinical scenario, are considered by the **EMSE** to be less important. However, the knowledge of these parameters are important for research work and for highly professional echocardiographers.

Left ventricular mass : automatically calculated by the machine during measuring LV conventional dimensions in the systole and in the diastole as shown in diagram 109.



Volume of LV = $(IVS + LVID_d + PW)^3$

Volume of Blood Pool = $(LVID_d)^3$

Myocardial Volume = LV Volume - Blood Pool Volume
$= (IVS + LVID_d + PW)^3 - LVID_d^3$

LV Mass = Myocardial Volume × 1.05 g/cm^3

Devereaux Regression:

LV Mass = $1.04\,[(IVS + PW + LVID_d)^3 - LVID_d^3] - 13.6g$

Diagram 109

Relative wall thickness :

Left ventricular hypertrophy is typically characterized as being concentric, eccentric, or physiologic, diagram 110,

Definitions :

1) Eccentric hypertrophy: Increase in left ventricular mass with chamber enlargement and relatively normal wall thickness.

Causes : regurgitant valvular lesions, i.e. volume overload

.

2) Concentric hypertrophy : Secondary to a predominant increase in wall thickness with normal chamber sizes,

Causes : pressure overload of systemic hypertension. i.e. pressure overload.

When evaluating patients for left ventricular hypertrophy, it is important to characterize the hypertrophy as being due to either **chamber enlargement** or **increased wall thickness.**

- One additional index of hypertrophy is relative wall thickness .

Relative wall thickness : posterior wall thickness + IVS thickness / LVEDD

Abnormal pathological left ventricular hypertrophy: Relative wall thickness > 0.45 .

3) Physiologic hypertrophy in highly trained athletes:

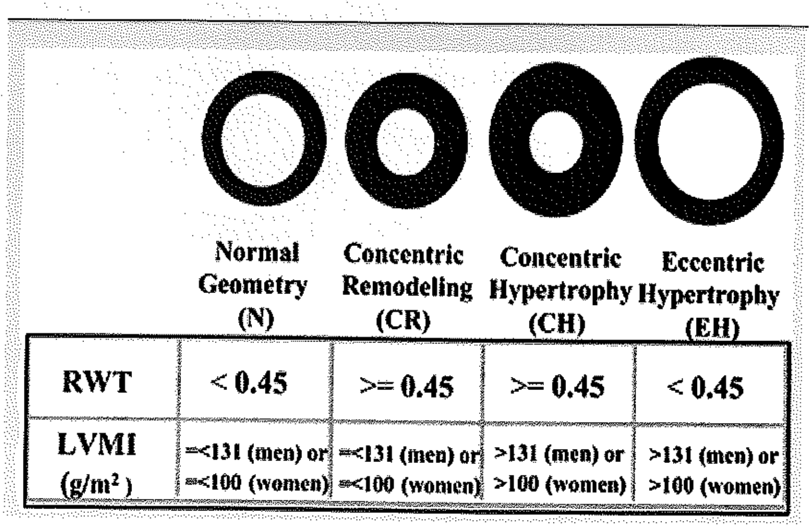
Slight increase in both wall thickness and chamber dimension.

Wall thickness more than 13 mm is unusual in physiologic hypertrophy.

Because the hypertrophy is a physiologic adaptation to physical training, wall stress tends to be normal.

The physiologic hypertrophy seen in athletes regresses relatively quickly after cessation of vigorous training.

It is exceptionally rare to see wall thickness more than 15 mm in the absence of significant hypertension, hypertrophic cardiomyopathy, or an infiltrative process.



	Normal Geometry (N)	Concentric Remodeling (CR)	Concentric Hypertrophy (CH)	Eccentric Hypertrophy (EH)
RWT	< 0.45	>= 0.45	>= 0.45	< 0.45
LVMI (g/m²)	=<131 (men) or =<100 (women)	=<131 (men) or =<100 (women)	>131 (men) or >100 (women)	>131 (men) or >100 (women)

Diagram 110 relative wall thickness.

Cross sectional area change : this is uncommonly used in clinical practice and is out of the scope of this book

Left ventricular dimensions:

2D echo and M.mode.

Left ventricular can be taken using conventional 2 D imaging as shown in diagrams 107 & 111.

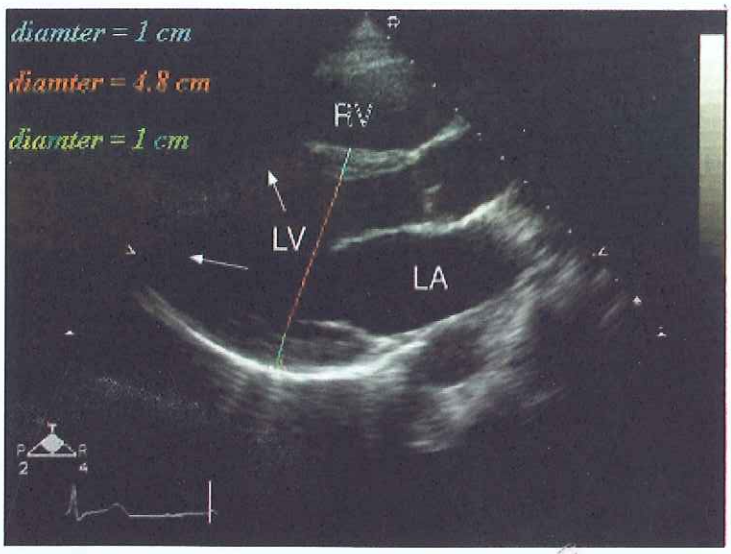
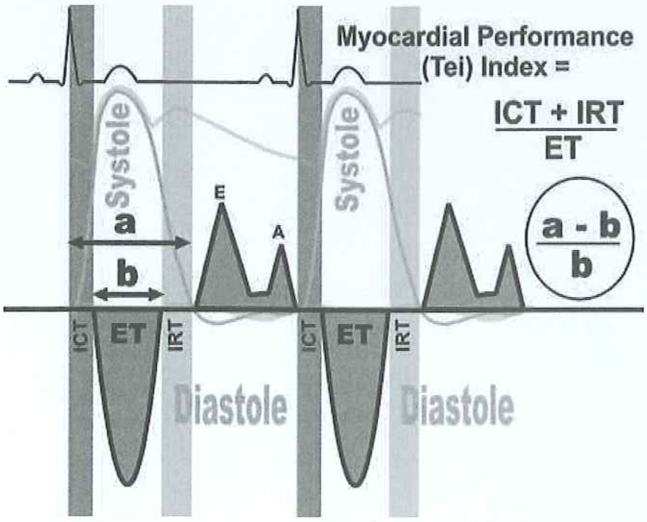


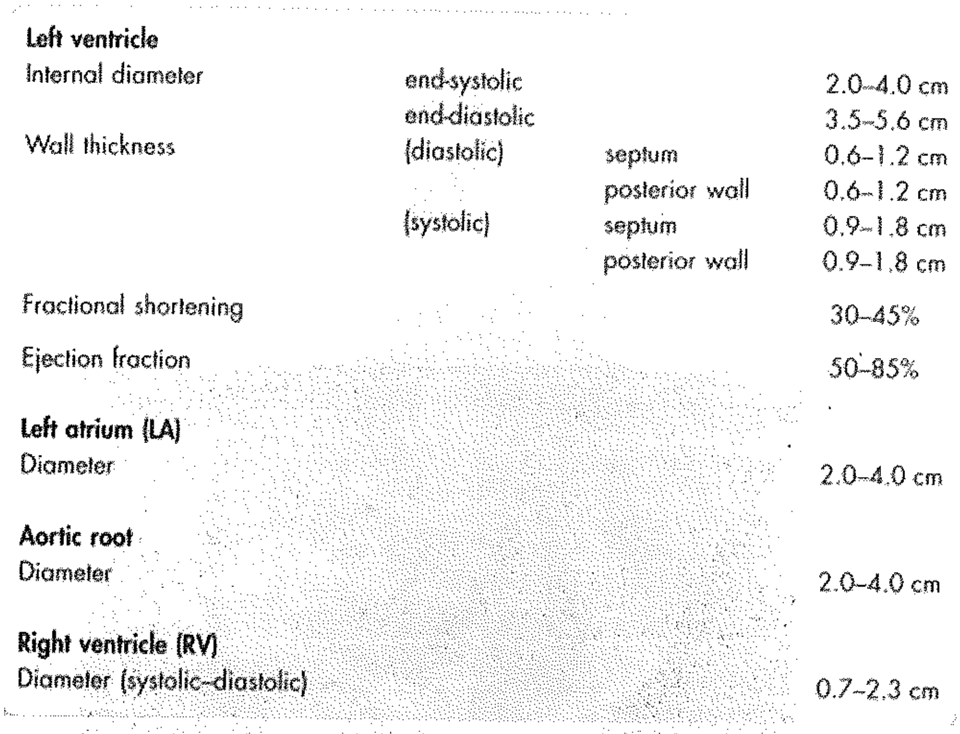
Diagram 111: Intracardiac dimensions.

Diagram illustrating the measurement of the myocardial performance index (MPI).

Normal MPI = 0.4

Poor systolic function is associated with larger MPI values.





Left ventricle			
Internal diameter	end-systolic		2.0–4.0 cm
	end-diastolic		3.5–5.6 cm
Wall thickness	(diastolic)	septum	0.6–1.2 cm
		posterior wall	0.6–1.2 cm
	(systolic)	septum	0.9–1.8 cm
		posterior wall	0.9–1.8 cm
Fractional shortening			30–45%
Ejection fraction			50–85%
Left atrium (LA)			
Diameter			2.0–4.0 cm
Aortic root			
Diameter			2.0–4.0 cm
Right ventricle (RV)			
Diameter (systolic–diastolic)			0.7–2.3 cm

N.B the definition of normal dimensions varies among different body surface area "BSA". So for border line dimensions &/or abnormal body surface area, correlating the dimension to the BSA is more accurate.

$$\text{BSA m}^2 = \sqrt{\frac{cm \cdot kg}{3600}}$$

Cm : patient's length in cm.

Kg : patient's weight in Kg.

Chapter 9: Assessment of the right ventricle:

In general the RV assessment by echo has many limitations owing to the normal non geometrical shape, heavily trabeculated walls, and most of it lies behind to the sternum.

When using any technique illustrated below always ensure that neither trabeculations nor a papillary muscle is included in the measurements.

RV can be assessed by all maneuvers like the LV.

There is a general consensus that the RV size and function are assessed qualitatively.

The **EMSE** advices that the quantitative measures should support the qualitative opinion.

2 D echo :

RV can be examined in all parasternal views. Also it can be assessed in the apical 4chamber view.

2 D measurements can be done as shown in diagram 112.

In general, in an apical 4 chamber view the RV should look triangular in shape and smaller than the LV. If the RV is the same size as the LV then it is moderately dilated, and if it is larger than the LV then it is severely dilated.

severe volume overload will lead to septum flatting forming what is called "D" shaped LV. Diagram 114

Abnormal RV walls can be looked for and reported. Diagram 115.

Also, the echocardiographer should search the RV for any abnormal masses and reports accordingly.

Simpson's method can also be applied, but is usually limited to research work. Diagram 112 B.

N.B take care to optimize the Apical 4 chamber view, because a foreshortened view will give a false impression of a dilated RV.

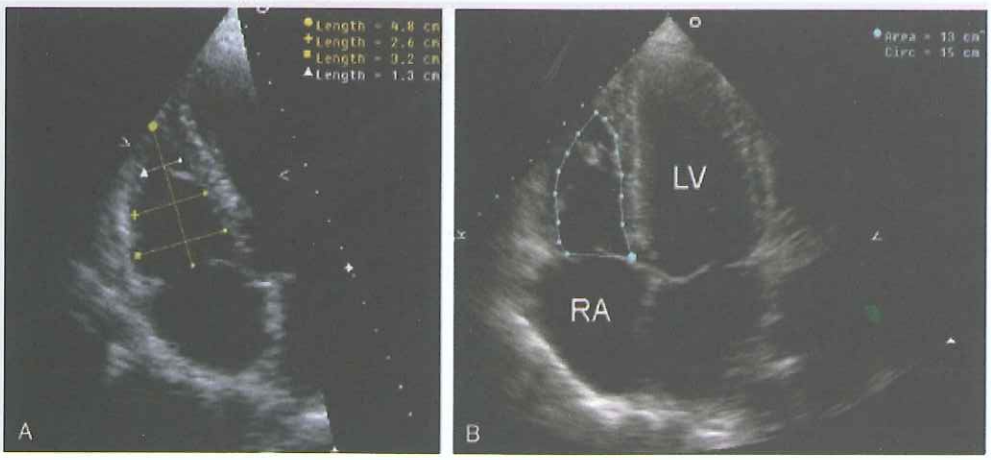


Diagram 112 :normal RV dimensions in apical 4 ch view.

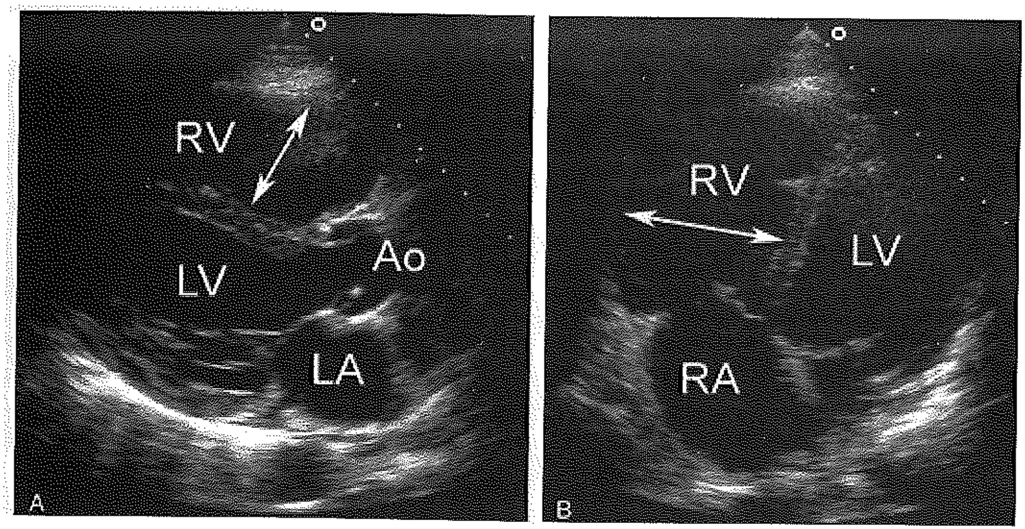


Diagram 113 : dialted RV in (A)PLAX and (B) Apical 4 ch views.

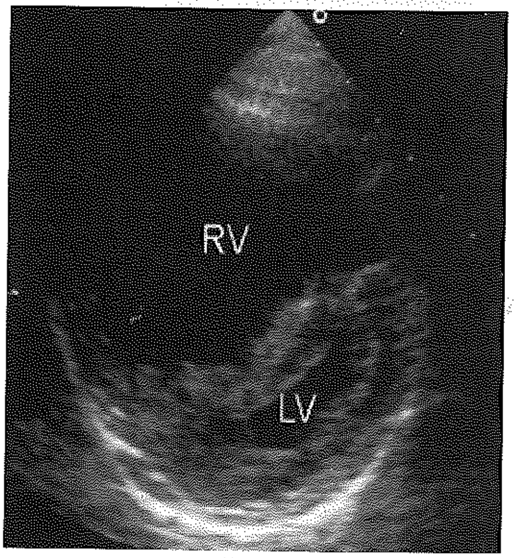


Diagram 114 : D shaped left ventricle secondary to it's compression by a huge RV in a case of severe pulmonary hypertension.

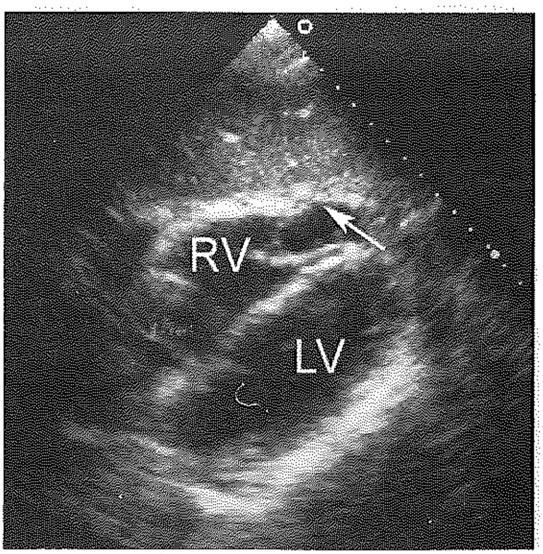


Diagram 115 : a subcostal view showing abnormal thickening and aneurysmal dilatation in the RV wall for differential diagnosis . However this case was diagnosed as Arrhythmogenic right ventricular dysplasia (ARVD)

Normal RV dimensions should not exceed :

2.5 cm in the PLAX view

3.3 cm in the Ap 4ch view basal cavity

3.5 cm in the Ap 4 ch view mid cavity

7.9 cm in the apical 4 ch view apex to base dimension.

M.mode :

In a PLAX view M.mode can be aligned and the RV walls can be measured as shown in the diagram 116.

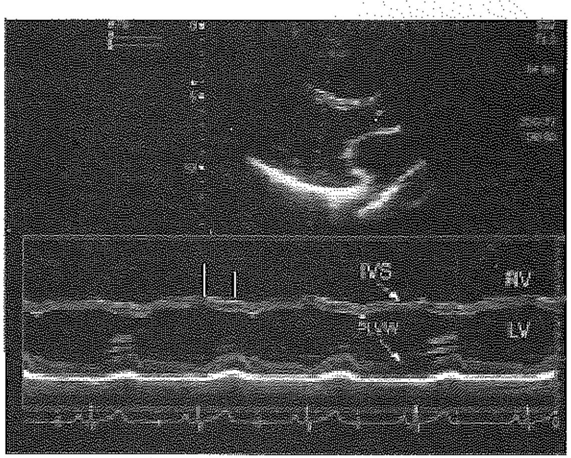
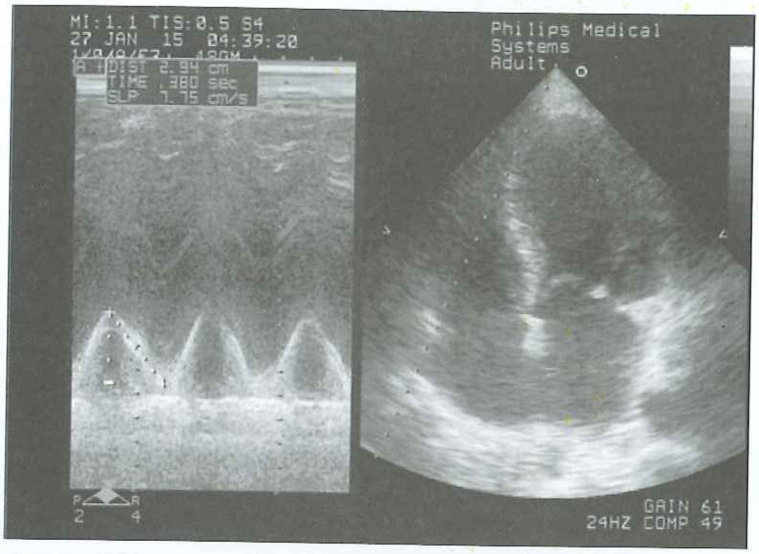


Diagram 116: RV measurment by M.mode in a PLAX view.

Tricuspid annular plan systolic excursion TAPSE :

With normal RV systolic function, the right ventricular base to apex systolic excursion will be relatively strong, by aligning the M.mode curser over the lateral annuals of the tricuspid valve, the magnitude of the excursion can be measured as shown in the diagram. Normal TAPSE > 1.5cm.

Diagram 117 for TAPSE



Ejection fraction and fractional shortening can be measured as the LV, but usually confined to research work.

Doppler U/S:

The same concept of measuring the LV stroke volume can be applied to the RV stroke volume. See diagram 118

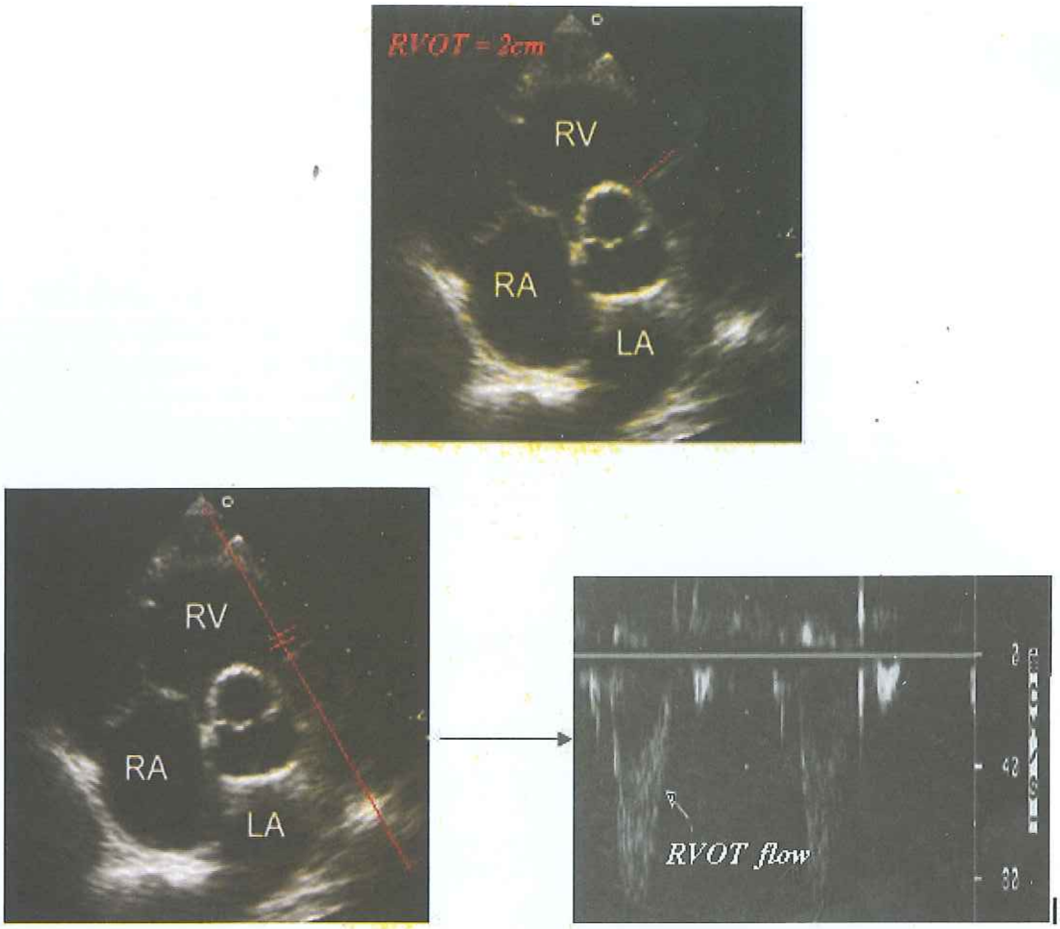


Diagram 118 : measuring the stroke volume for the RV.

RV stroke volume = RVOT cross sectional area {(RVOT diameter/2)^2 X π} **X** VTI RVOT (PW)

RV stroke volume = RVOT cross sectional area {(RVOT diameter/2)^2 X π} **X** VTI RVOT (PW)

Any abnormality found by any technique must be correlated to the various clinical conditions, for example, a dilated RV suggests volume over load (tricuspid or pulmonary regurgitation), a thickened wall suggests pressure overload (for example :Pulmonary stenosis or pulmonary hypertension).

Chapter 10: Assessment of the Diastolic function of the heart :

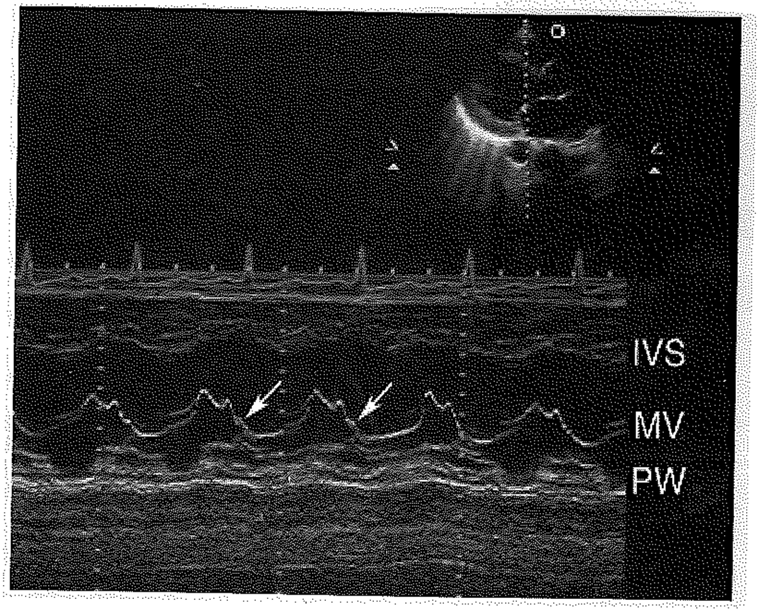
The main goal in the assessment o the diastolic function of the heart is to identify patients with heart failure with preserved ejection fraction HFpEF. These patients have the same mortality rate as those with systolic dysfunction, and during routine practice this group may be overlooked.

The main modality to diagnose diastolic dysfunction is the mitral inflow changes at the tips of the mitral valve elicited by the PW Doppler.

2 D echo :

Any abnormality seen by 2 D echo during the echocardiography examination will strongly suggest the presence of any of the various grades of diastolic dysfunction. For example, the presence of left ventricular hypertrophy, ischemic heart disease or moderate to severe valvular lesions is almost always associated with diastolic dysfunction.

M.mode:



A mitral valve echocardiogram demonstrating a B bump (arrows). These waves at the beginning of the CD line denote increased left ventricular diastolic pressure which confirms the presence of diastolic dysfunction.

Doppler:

1) **Pressure changes "at the tips of the mitral valve" between the LA and the LV serves as a good parameter to identify different grades of diastolic dysfunction :**

This is done by aligning PW Doppler with the sample volume placed at the tips of the mitral valve.

Normally the blood in the left atrium flow to the left ventricle by the suction force of the LV during the **e**arly filling phase i.e. passive filling phase . This leads to little contribution of the **LA** contraction in the diastolic phase, leading to the normal **E/A** ratio.

It is also very critical to remember that the left ventricular is; normally; highly compliant to the limit that it will accommodate any increase in the venous return, in a way that, if the a patient with normal LV diastolic function performed valslava maneuver, the increased venous return to the left side will be accommodated by the compliant LV and most of blood will passively flow to the LV, which is represented by the larger E wave, with less or equal amounts of blood left in the LA to be transmitted by the atrial contraction wave (the A wave). So, in normal individual, the E/A ratio does not reverse during valsalva maneuver. (unlike the pseudo normal pattern where the E/A ratio reverses during valsalva) see diagram 119.

grade I diastolic dysfunction:

In grade I diastolic dysfunction the LV compliance is poor (but the pressure is still normal). So the LV will not be compliant to accommodate the blood passively, and only little volume of blood will be transmitted by the E wave, leaving more volume of blood to be kicked by the LA, thus increasing the A wave. This leads to the final reversed E/A pattern see diagram 119.

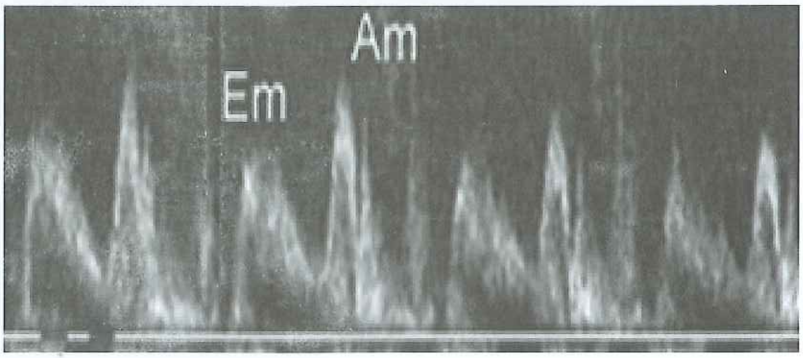


diagram 119 : reversed E/A ratio.

Grade II diastolic dysfunction :

With progressive deterioration of the diastolic function, the left ventricular pressure increases. This will lead to a pseudo normalization in the E/A ratio (one explanation is that the increased LV pressure will lead to a disproportional increase in the LA pressure and thus the blood will be pushed down to the LV with higher velocity, together with the decreased left atrial contraction owing to the left atrail myopathy, all this will end up with a larger E wave and a smaller A wave "pseudonormal")

The main technique to identify whether this E/A ratio is normal or not is to subject the LV to volume overload, this is done by valsalva maneuver that increases the venous return on the left side but

أي كمية blood هتوصل لل LV
هترفع الضغط العالي [illegible]

decreases it on the right side (revise physiology to understand and remeber the various stages of valsalva).

A normal LV will accommodate most of the increased venous return during the early filling phase, but an abnormal L.V will be unmasked in terms of its failure to accommodate the increased venous return during the early filling phase. This will be reflected as a smaller E wave and a larger A wave that occurs during the valsalva manoeuvre in cases of grade II diastolic dysfunction. See diagram 120.

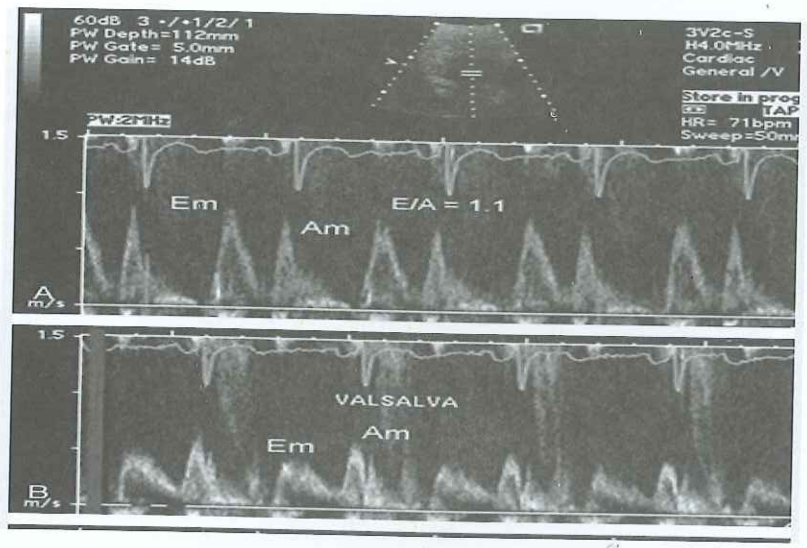


Diagram 120 : Grade II diastolic dysfunction. A: Normal E/A ratio, B: Reversed E/A ratio during valsalva,

Grade III diastolic dysfunction

In grade III diastolic dysfunction the LV pressure changes will appear as if the LV is surrounded by an iron shield. This is due to a restrictive or constrictive pathology.

In this situation, there is no LV compliance to accommodate the early filling blood flow, so the E wave velocity peaks, then there is no compliance to maintain the LV pressure from abrupt falling, leading to a short deceleration time (and subsequently a short pressure half time). In this grade of diastolic dysfunction the left atrial myopathy is markedly increased leading to a very poor atrial kick represented by a small "A" wave. See diagram 121.

أي حجم blood
هتفرغ الـ LV
هتفرغ الـ pr.
زيادة جامدة.

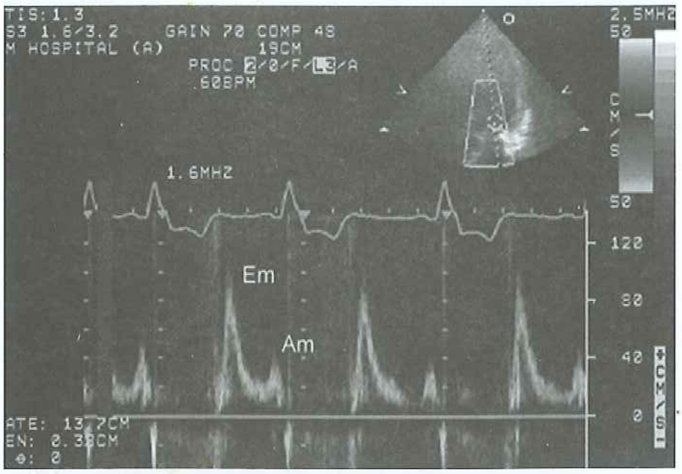


Diagram 121 : Grade III diastolic dysfunction.

N.B : Grade IV diastolic dysfunction is defined by the irreversible pattern (fixed E/A ratio) of grade III during valsalva. If the E/A ratio is reversed during valsalva then the grade III diastolic dysfunction is diagnosed, if it is not reversed then the diastolic dysfunction is grade IV.

Summary of diastolic dysfunction: Diagram 122 A & B :

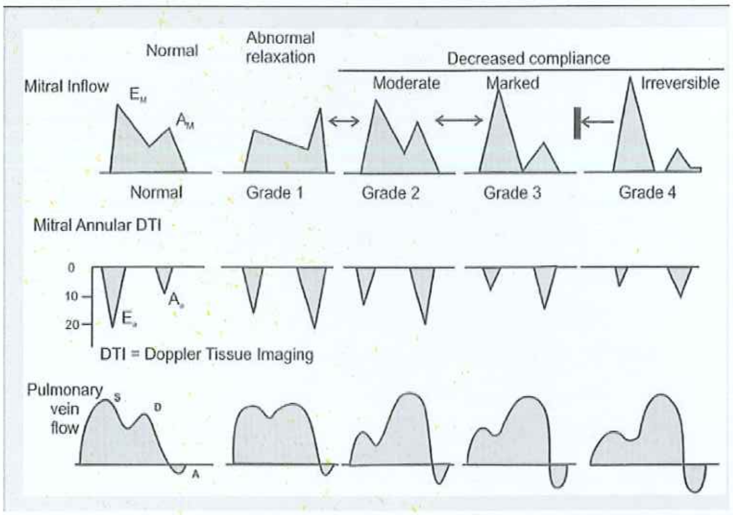
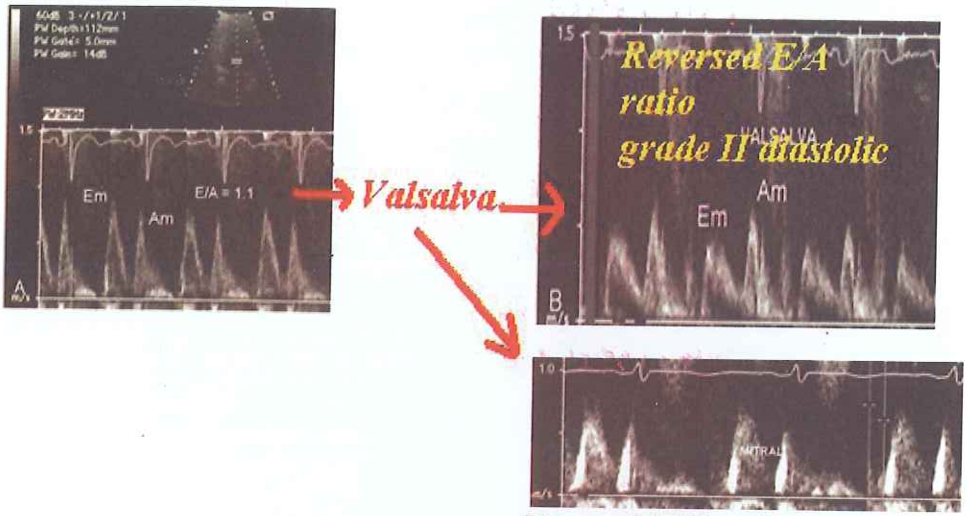


Diagram 122 A

Diagram 122 B



Normal E/A ratio--> normal diastolic function

EMSE clinical tip :

In the presence of left ventricular abnormality, the normal E/A ratio is mostly in fact a pseudo normal one, i.e. grade II diastolic dysfunction.

Pulmonary venous flow to assess the diastolic function:

Attributed to the difficult feasibility to image a pulmonary vein in an adult individual, the assessment of diastolic dysfunction from pulmonary venous flow is only used when mandatory.

- pulsed Doppler techniques used for determining mitral inflow can be used to determine the pattern of pulmonary vein flow into the left atrium. Diagram 123
- Typically, imaging is performed from the apical view.

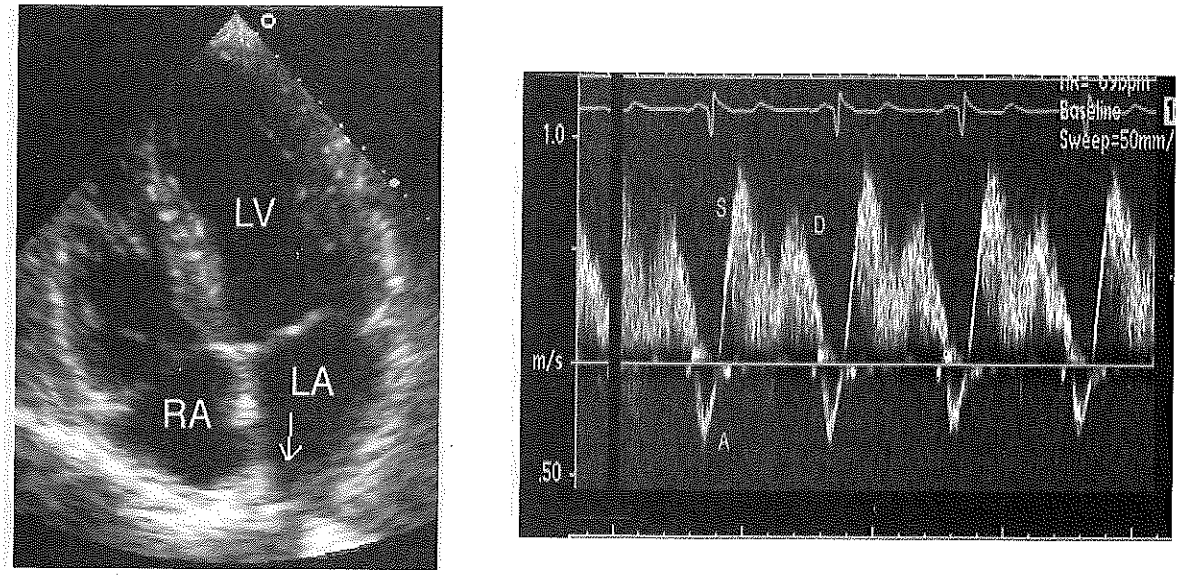
The sample volume can then be placed at the orifice of the pulmonary vein and pulsed wave Doppler imaging is used to record the flow envelope.

- Normal pulmonary vein inflow consists of a diastolic and systolic phase as well as atrial reversal. In some instances, there will be two distinct systolic flow phases.

In normal, disease-free individuals with normal diastolic properties of the ventricle, the velocity of systolic flow typically equals or exceeds that of diastolic flow.

- Atrial regurgitation wave : For patients who are in sinus rhythm, there is a short, low-velocity reversal of flow coincident with atrial systole, the so-called pulmonary vein A-wave.
- With increasing stiffness and decreasing compliance of the left ventricle, emptying of the left atrium becomes incomplete during diastole. Because of this, at the onset of ventricular systole, left atrial volume and pressure are elevated.

This results in a reduction of flow volume into the left atrium (from the pulmonary veins) during ventricular systole, which is reflected in a blunting of the systolic flow and a relative increase in the diastolic flow (diagram 124) because the overwhelming majority of flow out of the pulmonary veins occurs during ventricular diastole coincident with emptying of the left atrium. The incomplete emptying and increase in atrial pressure also result in an exaggeration of both the velocity and width of the pulmonary vein A-wave. The pulmonary vein A-wave duration can be compared with the mitral A-wave duration. At least one study has suggested that, if the pulmonary vein A-wave duration exceeds the mitral A-wave duration, the left ventricular end-diastolic pressure exceeds 15 mm Hg in the majority of cases.



The white arrow is pointing to one of the pulmonary veins, PW sample volume can be placed inside the pulmonary vein to detect the S D waves.

Diagram 123 : Pulmonary vein pulsed Doppler imaging showing normal diastolic function. Note the systolic predominance of forward flow from the pulmonary veins and relatively narrow pulmonary vein A-wave. Also note the indistinct nature of the pulmonary vein A-wave .

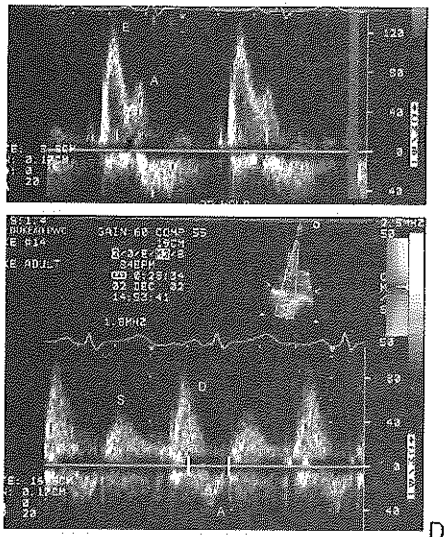


Diagram 124 : Pulsed Doppler recording of mitral inflow (top) and pulmonary vein flow (bottom). Note in this example the reduced S wave of the pulmonary vein, suggesting diastolic dysfunction. Note also the prominent wide pulmonary vein A-wave.

TDI to assess the diastolic function:

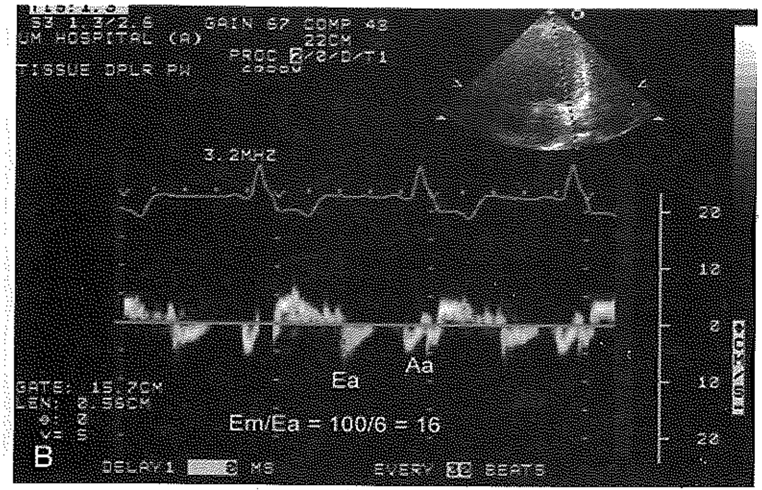


Diagram 125 : Tissue Doppler over the lateral annuals of the mitral valve.

Remember that during systole the base of the heart moves towards the apex, i.e. towards the probe, so it will give a positive systolic deflection wave S'. During diastole the base of the heart moves away from the apex, i.e. away from the probe, so it will give two negative deflections E' and A'which are equivalent to the E/A ratio of the classic mitral filling, the A' will be lost in cases of Atrial fibrillation.

Normally the systole should be powerful enough to eject the blood out of the LV, so, normally the S' should be of a large value. Normal S' > 7.5 cm/s. decreased S' denotes systolic dysfunction.

Normally the diastole should be of good compliance, which means that the base should move away from the base freely and with high velocity. So, normally the E' should be of a large value. Normal E' > 12 cm/s.

Normal E/E' ratio should be less than 8

N.B : E' correlates with the LV diastolic function

E/E' correlates with LVED pressure, so ↑ E/E' ratio denotes ↑ LVED pressure.

Chapter 11 : Cardiomyopathies :

The EMSE classification of cardiomyopathies :

1) Cardiomyopathies with specific echocardiographic findings :

a) **Noncompacted myocardium**

b) **Infiltrative causes :**

**Hemachromatosis*

* *Amyloidosis*

* Glycogen storage diseases

c) **Hypertrophic cardiomyopathies**
  *Asymmetric septal hypertrophy (idiopathic hypertrophic cardiomyopathy
  *Obstructive vs. nonobstructive
  *Concentric hypertrophic cardiomyopathy
  *Isolated apical hypertrophic cardiomyopathy
  *Atypical hypertrophic cardiomyopathy
d) **Hypertensive cardiomyopathy**
e) **Valvular cardiomyopathy**
f) **Ischemic cardiomyopathy**

2) Cardiomyopathies without specific echocardiographic findings :

a) **Dilated cardiomyopathy**
b) **Idiopathic cardiomyopathy**
c) **Familial cardiomyopathy**
d) **Postpartum cardiomyopathy**
e) **Infectious**
  * Postviral myocarditis
  * Human immunodeficiency virus related
  **Legionella* infection
  *Sepsis (gram negative)
f) **Toxic cardiomyopathy**
  *Adriamycin
  *Alcohol
  *Carbon monoxide poisoning
  *Other chemotherapy
g) **High-output cardiomyopathies**
  *Tachycardia-mediated cardiomyopathy
  *Thyrotoxicosis

*Nutritional (beriberi, thiamine deficiency)
*Peripheral left-to-right shunt lesions
*Anemia

h) Restrictive cardiomyopathy

* Idiopathic
*Post-radiation therapy
*Endocardial fibroelastosis

i) Other

* Friedreich ataxia
*Muscular dystrophies

Most of cardiomyopathies share the same echocardiographic criteria in terms of:

1) Poor ejection fraction,
2) Diastolic dysfunction
3) Dilated left ventricle in most instances.

It is important to warn that at the end stage, all cardiomyopathies may be indistinguishable from each other, and we can only suggest a cause from either the clinical history or an irreversible effect that caused the disease (ex. Rheumatic heart diseases).

However some cardiomyopathies have unique echocardiographic features than others.

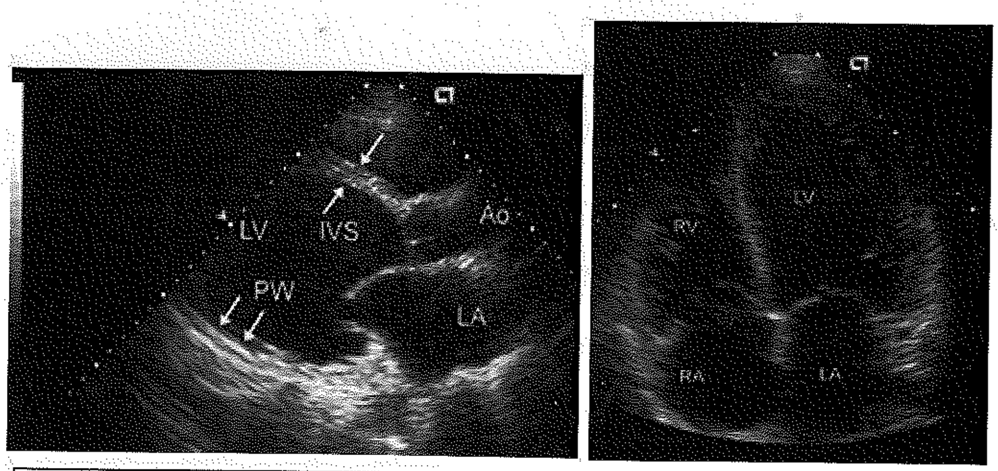
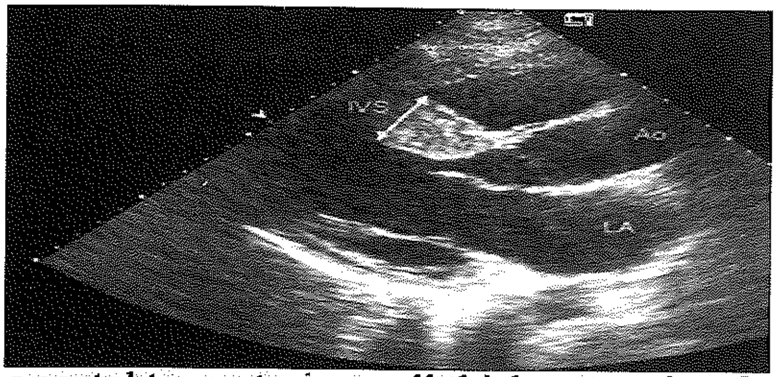


Diagram 126: dilated LV, thinned out left ventricular walls in PLAX and apical 4 ch views.



a septal to posterior wall thickness ratio of 1.3:1is considered evidence of inappropriate septal hypertrophy that is associated with HCM and HOCM

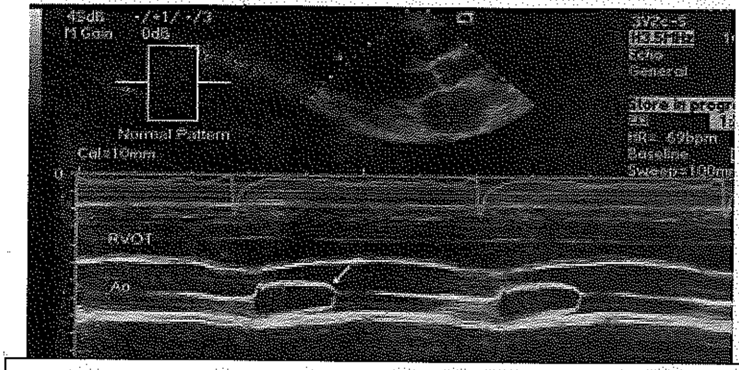
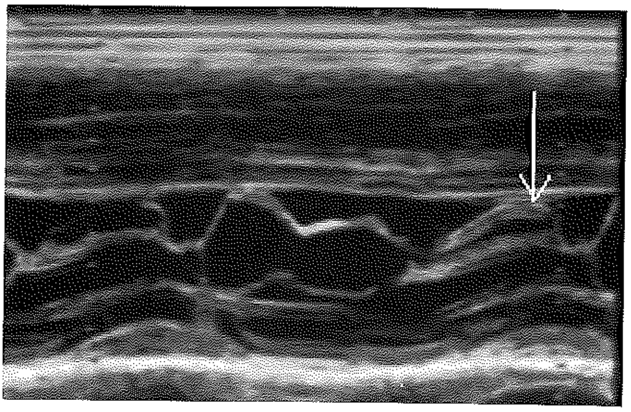


Diagram 127 : Poor systolic excursion of the aortic valve, notice the rounded closure shape of the AV in the M.mode .

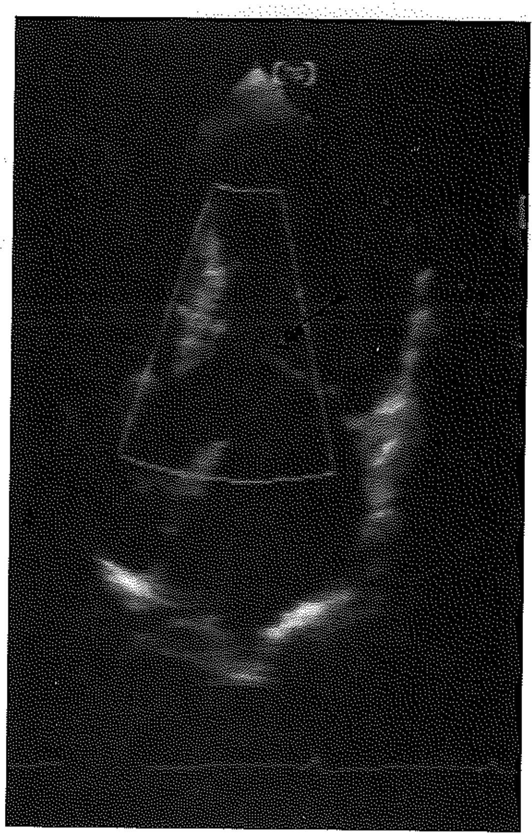
Review over the echocardiographic findings for the cardiomyopathies with specific echocardiographic criteria :

Diagram 128 A : a case of hypertrophic obstructive cardiomyopathy. Systolic anterior motion of the mitral valve in M.mode trace, shown by the arrow.



N.B: To diagnose SAM (systolic anterior motion of the mitral valve) the anteriorly displaced "CD" line of the mitral valve must reach back to the base line at the D point before the beginning of the diastole.

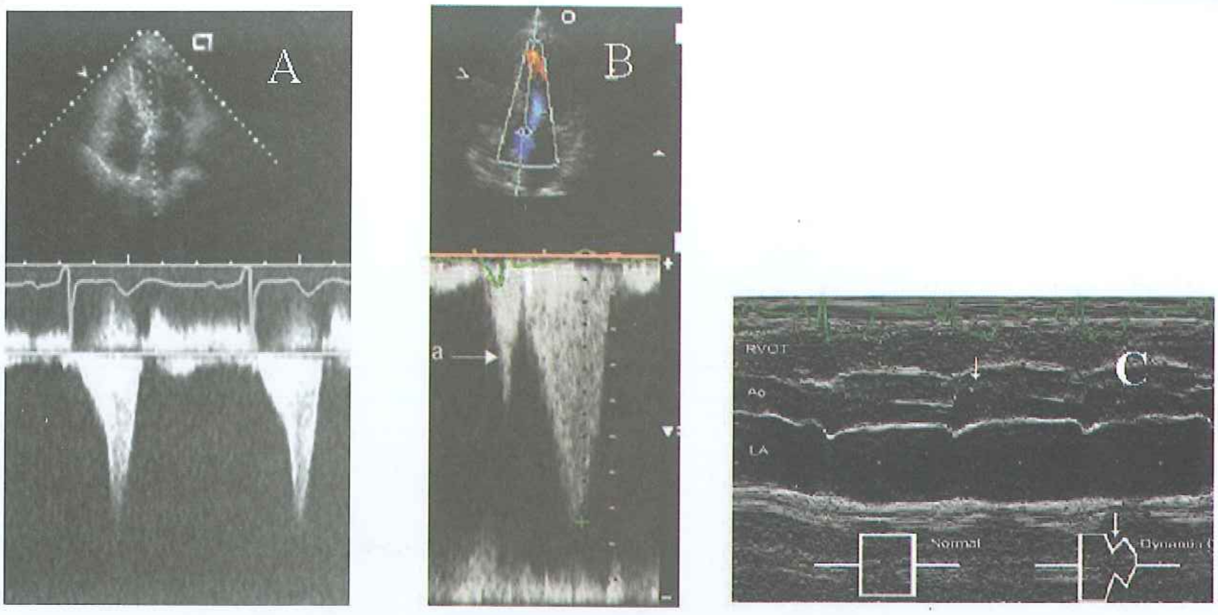
Diagram 128 B : notice the sucked mitral valve cooptation zone towards the LVOT as shown by the arrow.



128 B :

Two theories had been postulated to explain the SAM, the first depends on the venturi effect, where any high velocity jet originating at the LVOT creates vacuum that sucks the mitral valve.

The second theory depends on the fact that the fibrous tissue trigone of the heart is weak in cases of HOCM or HCM, so the mitral valve is squeezed into the LVOT during the systole, this theory explains the presence of SAM in HCM.



Different echocardiographic findings in HOCM in the Doppler study and M.mode trace.

A: Notice the dagger shaped flow due to the late systolic peaking that occurs in HOCM

B: Note the prominent presystolic flow in the outflow tract (a) due to atrial contraction against a highly noncompliant and hypertrophied left ventricle.

C: demonstrates systolic notching of the aortic valve (arrow)in the M.mode. Normal closure is schematized in the inset.

Diagram 129: Non compaction cardiomuopathy

Notice the noncompacted areas of myocardium shown by arrow.

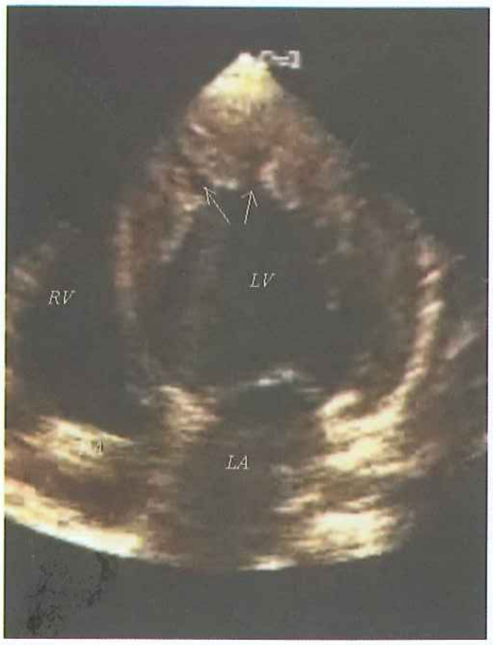


Diagram 130 : infiltrative disease of the heart for differential diagnosis.

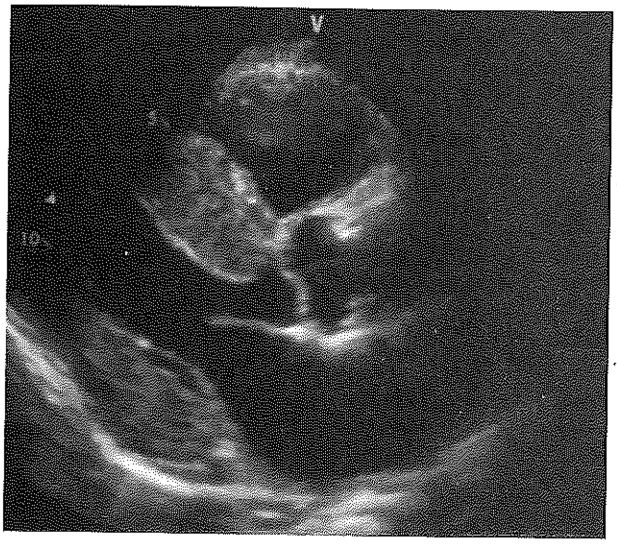
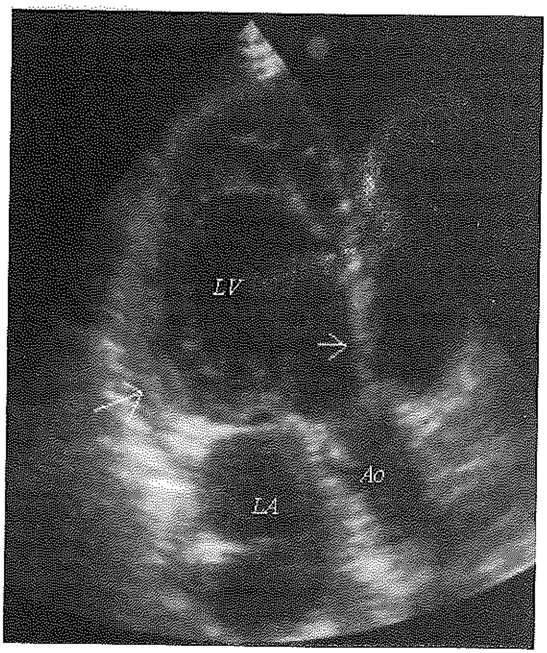


Diagram 131: Ischemic cardiomyopathy

Apical 3 ch view, Notice the thinned out septum, while the other walls are normal.

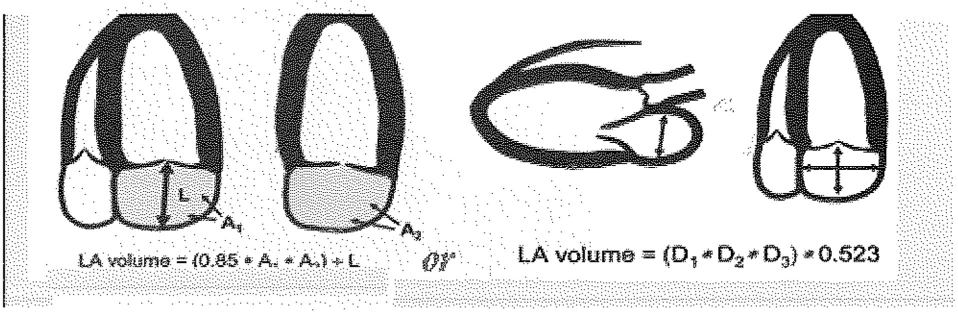


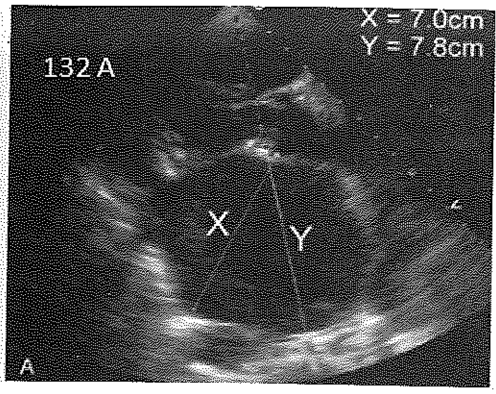
Chapter 12 : Assessment of the left atrium

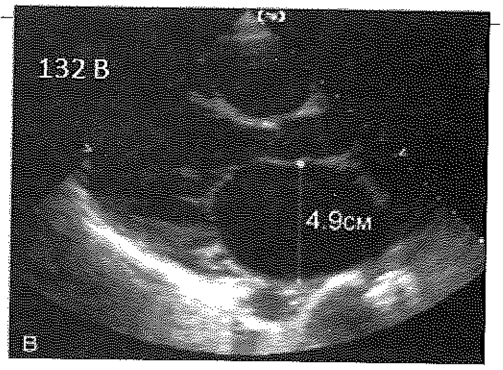
2D echo :

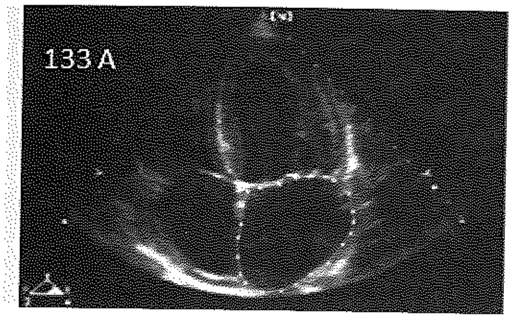
In any view optimizing the left atrium, it can be measured at its end diastole, as length and width using the classic linear measurements as shown in diagram 132 A&B, or by using the area trace method as shown in diagram 133 A&B.
The sonographer must look for any other abnormalities in the LA, mainly left atrial masses, for example myxoma, thrombus, vegetation. And any mass can be measured in two different dimensions. A full discussion is made in "intra cardiac masses".
Uncommonly the warfarin ridge may be seen in LA between the left atrial appendage and the left upper pulmonary vein. (This normal mass is classically seen by TEE and in some instances in TTE. This ridge is the remnants of the left sided embryological SVC)

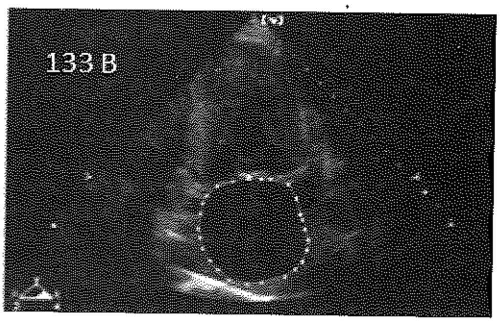
Estimation of the Left atrial volume :
The left atrial volume correlates better with the MRI derived volumes than the simple two dimensional measure.


L
A_1
A_2
LA volume = $(0.85 * A_1 * A_2) \div L$
or
LA volume = $(D_1 * D_2 * D_3) * 0.523$

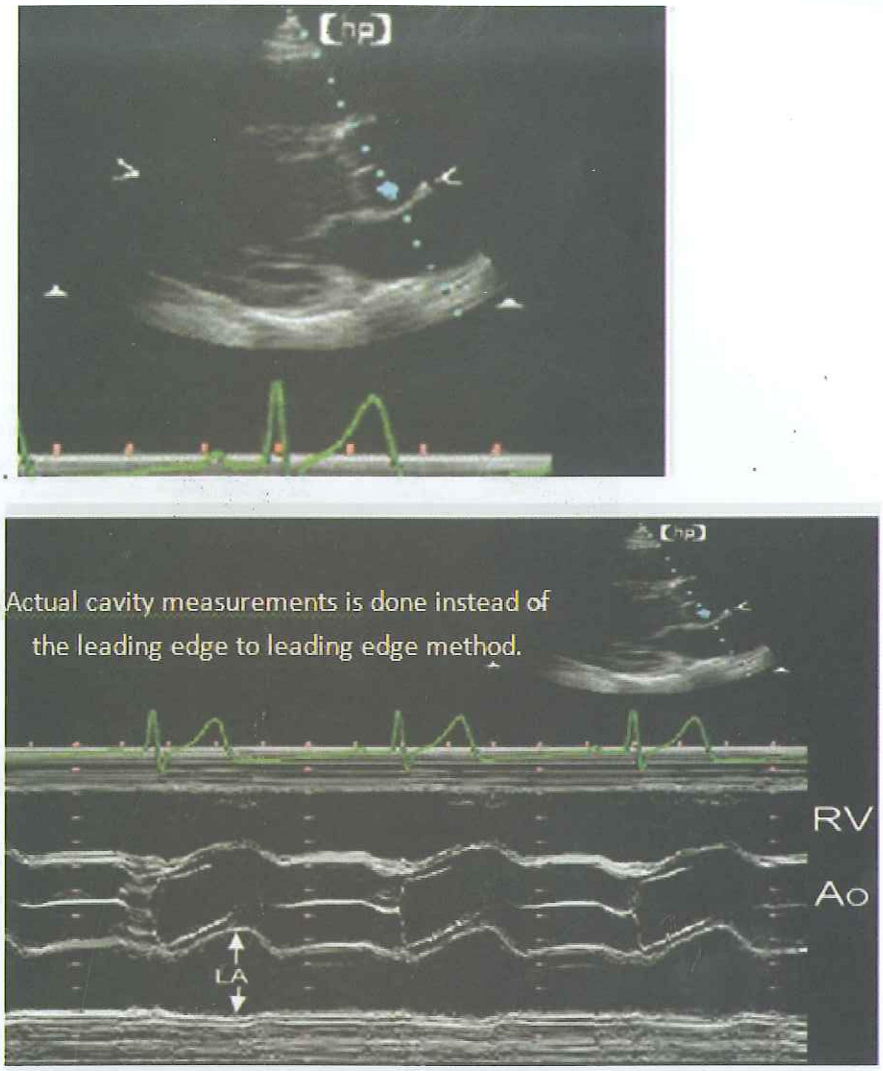








M.mode: In the PLAX view M.mode can be aligined and the LA can be measured from the M.mode trace. See diagram 134



Actual cavity measurements is done instead of the leading edge to leading edge method.

Diagram 134: LA measurement by M.mode.

There are 2 ways to identify the left atrial layers:

1) The classic school: leading edge to leading edge method.
2) The Actual *tissue blood interface* i.e. "the actual cavity"

The **EMSE** recommends to measure the actual cavity (as shown in diagram 134) rather than the leading edge to leading edge method

Doppler:

Either C.W Doppler or color Doppler can be used to see if any high velocity jets are filling the LA, for example, a high velocity jet of mitral regurgitation can be seen filling the LA, or a right to left shunt can be seen. However another uncommon clinical manifestation that may be seen is a high velocity jet flowing from the orifice of the pulmonary vein denoting pulmonary vein stenosis. See diagram 135.

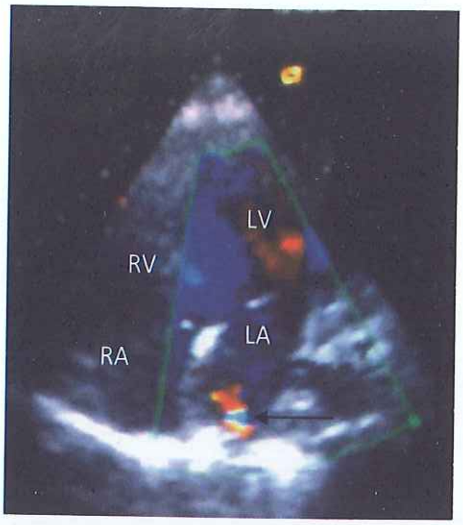


Diagram 135 : Notice the high velocity jet (denoted by arrow) arising from the pulmonary vein, denoting pulmonary vein stenosis.

N.B in cases of more than mild mitral regurgitation, the left atrium will almost always dilate, except in acute mitral regurgitation, however in the latter the clinical status of the patient will suggest an acute cause.

Common Causes of LA dilatation : IHD, MR, HF & HFpEF, AF.

Normal LA dimensions : <4 cm

mildly dilated: 4-4.5, moderately dilated: 4.5- 5, severely dilated : > 5 cm.

Chapter 13 : assessment of the Right atrium :

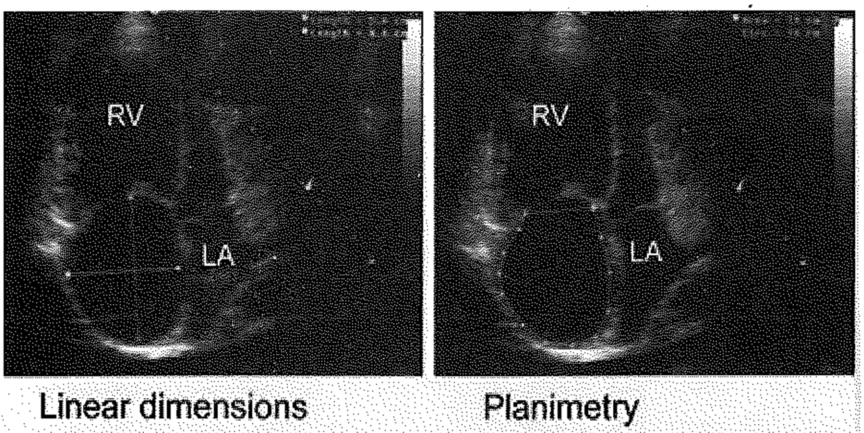
In general the RA can be assessed exactly like the LA, keeping in mind

1) The different venous flow pattern. See diagram 143 A.
2) The normal masses that can be seen in the right atrium.

2D echo :

The 2 D measurements are most commonly taken in the apical 4 chamber view. See diagram 136 .

In general the RA should not exceed the LA size.



Linear dimensions

Planimetry

Diagram 136: RA measurements.

Normal masses in the RA :

All the normal masses that can be seen in the RA may be misdiagnosed as pathological lesions, so, the sonograher's experience and correlating the finding to the clinical condition plays a critical role in such situations.

1)A remnant Eustachian valve can be seen at the IVC opening and identified on the anatomical basis near the origin of the coronary sinus. See diagram 137 &138 (some times it is mobile but less mobile than the chiari network).

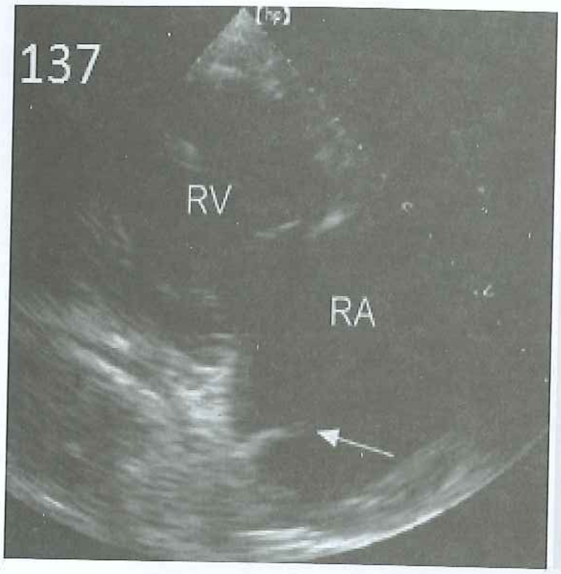
Origin : The eustachain valve is an embryological remnant that shunted the blood from the IVC to the LA in the embyriological circulation.

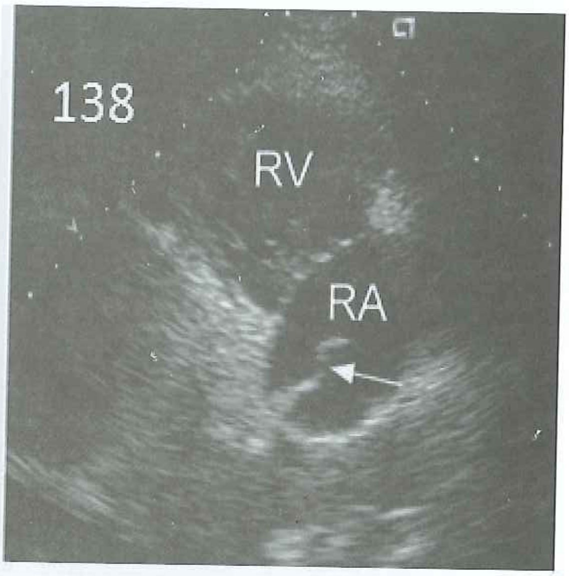
2)A chiari net work can also be seen, however it does not have a fixed attatchment site. See diagram 139.

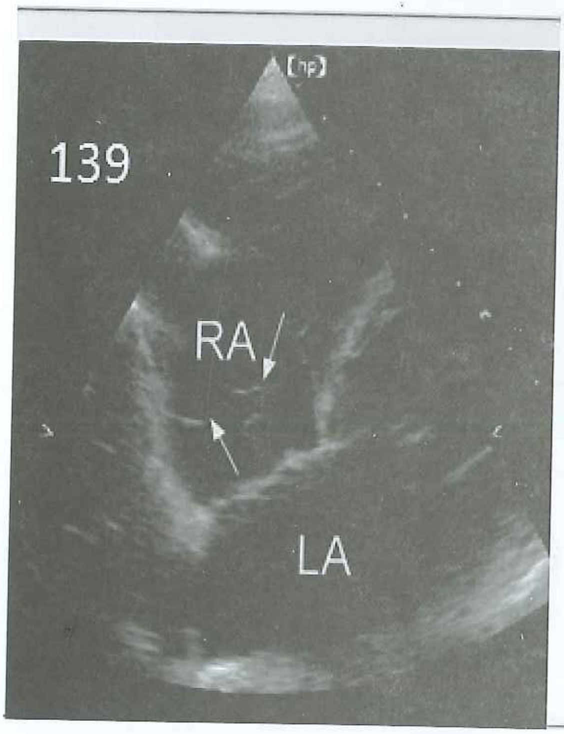
Origin : A chairi net work is a trabeculea remenet from the SVC/ IVC opening into the RA.

3)Crista terminals may be encountered especially in a bicaval view. See diagram 140.

Origin : Crista terminalis is the site of fusion between the smooth and rough parts of the RA. See diagram 140 .







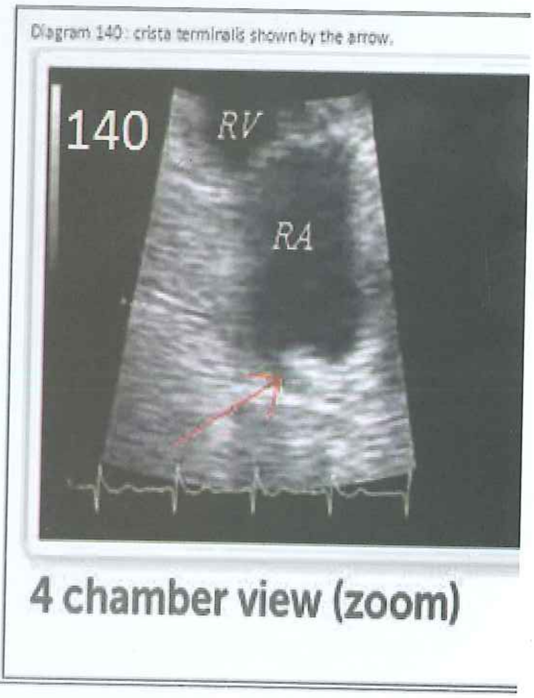


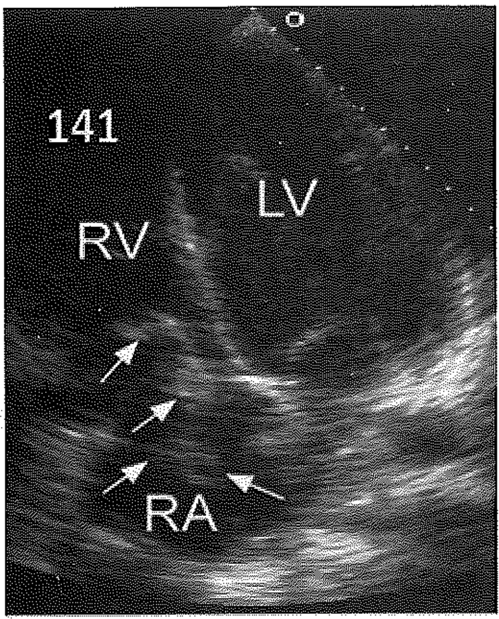
Diagram 140 : crista terminalis shown by the arrow.

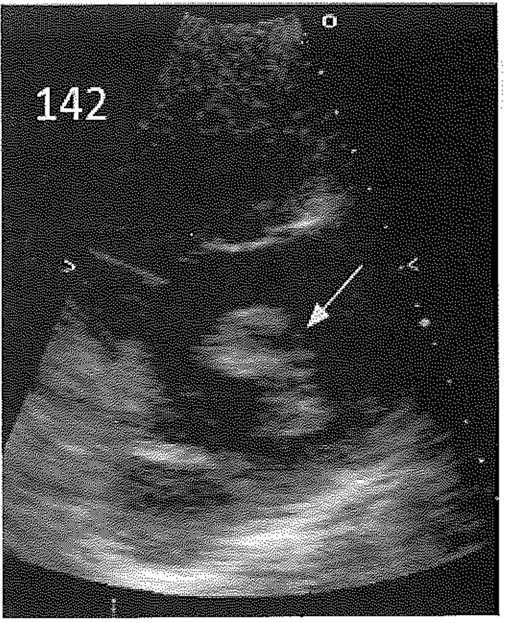
4 chamber view (zoom)

Exampels for abnormal RA masses:

1) Right atrial thrombus,
2) Vegitation.
3) Iintracardiac tumor (see intra cardiac masses)

Exampels are given for RA thrombus in diagrams 141 (apical 4 ch view) & 142 (parasternal inflow flow.)





M.mode :

Assessment of the RA by M.mode is uncommon in clinical practice, however an anatomical M.mode (M.mode steer) may be aligned over the RA if clinically relevant.

Color Doppler:

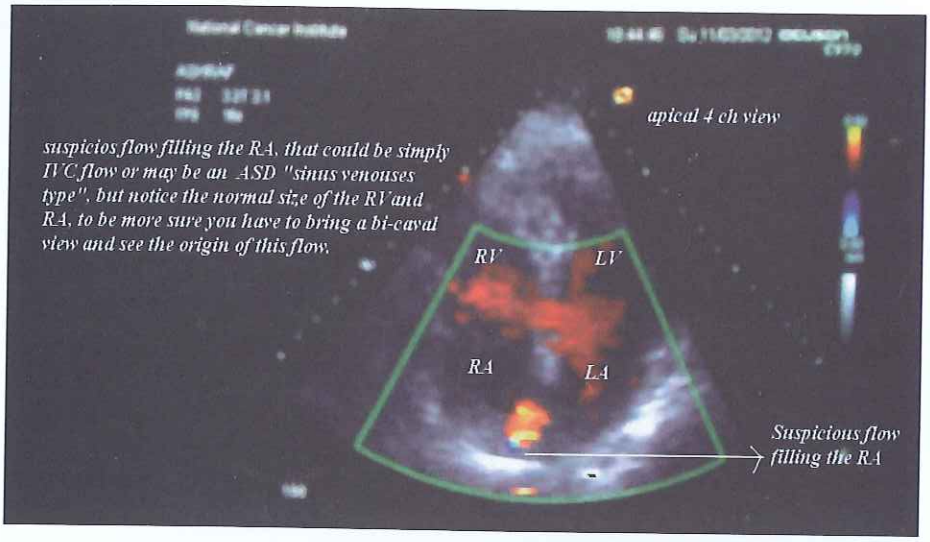
Normally no high velocity jets are supposed to be seen filling the RA.

Abnormale high velocity jets that can flow into the RA includes:

1)Tricuspid regurgitation (N.B : mild TR with no heamodynamic effect is a normal finding,see tricuspid regurgitation)

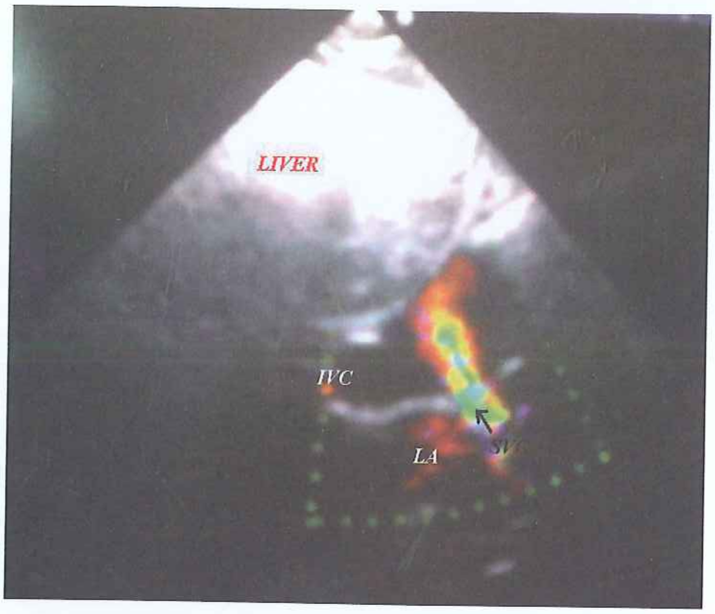
2) Left to right shunt ASD.(see assessment of IAS)

3)Exaggerated IVC/ SVC flow into the RA. See diagram.



suspicios flow filling the RA, that could be simply IVC flow or may be an ASD "sinus venouses type", but notice the normal size of the RV and RA, to be more sure you have to bring a bi-caval view and see the origin of this flow.

Diagram 143 A : A suspected flow filling the RA in the apical 4 chamber view.



143 B : The same patient shown above, the flow is proved to be arising from the SVC flow in the bicaval view, i.e. normal venous return.

This exaggerated flow from the SVC may sometimes raise unnecessarily suspicions about the presence of a sinus venouses ASD.

A precise differentiation between the sinus venousus ASD flow and the exaggerated SVC flow can be done from a bicaval view. See diagram 143 A&B.

Also the presence of the heamodynamic effect of an ASD; typically in the form of pulmonary artery, RA and RV dilatation; raises the possibility that the query flow is due an ASD.

EMSE clinical insights of the Importance of the assessment of the left atrium and right atrium :

Although the atrial size and function does not affect the treatment plane (except in minority of patients or in cases of hugely dilated atria), their assessment is still very important as an abnormal atria will direct our attention for an underlying cardiac abnormality or anomaly.

A dilated left atrium may be an important clue for the diagnosis of HFpEF. In this situation the LA will be considerably dilated with a normal E/A ratio, directing the attention for a pseudo-normal pattern.

Also a dilated LA may be an important clue for a VSD or a PDA. Sometimes a VSD is difficult to be seen especially the apical and marginal VSDs. A PDA in an adult may be missed, but their heamodynamic effects; in terms of LA, LV and PA dilatation; should not be overlooked.

A dilated RA can be the first clue for an ASD coronary sinus defect, where there is no actual defect in the interatrail septum. (assessment of the IAS)

Also a dilated RA can be an important clue to diagnose partial anomalies pulmonary venous return.

Dilated RA serves as an important parameter to asses and reassess the pulmonary artery pressure. For example : if the sonographer measured the pulmonary artery pressure within normal or mildly elevated but he is seeing a moderately to severely dilated RA, he must reevaluate the PAP many times in order to ensure the estimated measure of the PAP whether it is true or it is underestimated.

Chapter 14: Assessment of the intra cardiac septa and shunt lesions:

EMSE clinical insights on the intra cardiac shunts:

The hidden shunts : 1)ASD coronary sinus type 2) ASD sinus venosus defect 3)PAPVR, 4)apical VSDs.

It was found that some shunt lesions are not obvious during the routine studies, for example an ASD coronary sinus type or a PAPVR, these shunts must be suspected first in order to be diagnosed. Suspicion will be made on the basis of their well known heamodynamic effects. Another example is a marginal or apical VSD where the sensitivity of color Doppler is as low as 50% to detect this lesion and the diagnosis may be missed unless suspicion was raised towards it, again, in terms of the heamodynamic effect.

Also a lot of surgical decisions will be made depending on the heamodynamic effect of the lesion.

So the **EMSE** recommends that: the **Effect of the lesion** "in special circumstances" is **more important than the defect.**

So a detailed understanding of the heamodynamics is mandatory during the assessment of the intra cardiac septea.

Classic Heamodynamics of shunt lesions :

All shunt lesions will cause chamber dilatation due to the increased blood flow caused by the specific shunt, but for sure the chamber dilatation will differ according to the shunt type.

The main factor that determines chamber dilatation is the timing during which the increased flow is shunted to this chamber. If the timing of the increased flow is shunted during systole then the chamber will not dilate. But if the timing of the increased flow is received during diastole then the chamber will dilate. The great vessels do not apply for that rule, and they will dilate whenever they are subjected to an either increased pressure or volume overload.

1)VSD:

In a VSD the blood is shunted from the LV to the R.V " during systole" so the RV will not dilate (because it will be systolic i.e. contracted and stiff) leaving no chance to dilate by volume overload as the blood will be immediately shunted to the elastic pulmonary artery that will dilate by both volume and pressure over load. (The pulmonary artery now receives an increased blood volume and is subjected to the high LV pressure). This increased volume and pressure will be transmitted to the lung (lung plethora and pulmonary hypertension) then to the LA. Now the LA will receive an increased blood volume during its filling phase i.e during diastole so the LA will dilate by volume over load, then this increased volume will be transmitted to the LV "during diastole" . The L.V will also dilate by volume over load, now the blood will be unequally divided during the systole via two pathways, i.e. via the aorta and the VSD, so the ascending aorta will not dilate (unlike PDA where the ascending aorta will dilate as the increased volume will have only one pathway to leave the LV, which is the aorta).

To summarize, the VSD causes pulmonary artery, LA and LV dilatation.

Pulmonary A / HTN
+
Lt side
No aorta

Pulmonary
HTN

PDA :

In PDA the blood will be transmitted from the descending aorta to the left pulmonary artery (remember that the LPA, RPA and the main pulmonary artery is one chamber), so the pulmonary artery will dilate by both volume and pressure overload, this increased volume and pressure will be transmitted to the lung (lung plethora and PHT++). Then the increased volume will be transmitted to the LA during its filling phase causing its dilatation by volume over load, then to the LV during the diastole leading to LV dilatation by volume over load. Now the increased volume has only one passage to be ejected from, which is the ascending aorta, causing its dilatation. (unlike the VSD where the increased volume of blood in the LV will be ejected via the aorta and the VSD so the aorta will not dilate in VSD).

To summarize, PDA will lead to pulmonary artery, LV, LA and ascending aortic dilatation.

P.HTN

Pul. < artery
HTN
+
Lt side
+ Aorta

ASD:

In an ASD the blood will flow from the LA to the RA. The key point to understand this shunt is to remember that it is a continuous shunt (as the LA pressure is continuously higher than the RA pressure). Now the RA will dilate by volume overload, then the increased blood volume will be transmitted to the RV during diastole causing its dilatation. Then the increased blood will be transmitted via the pulmonary artery causing its dilation by only volume overload. The increased volume will be transmitted to the lung (lung plethora and later on PHT++), then the increased volume will be transmitted to the LA during diastole, ***BUT REMEMBER*** that the ASD is a continuous shunt, so the increased blood volume received by the LA will simply displace the blood already present in the LA to the low pressure RA via the shunt, and so, LA be will neither be subjected to volume overload nor will it be dilated.

To summarize, ASD will lead to pulmonary artery RA and RV dilatation.

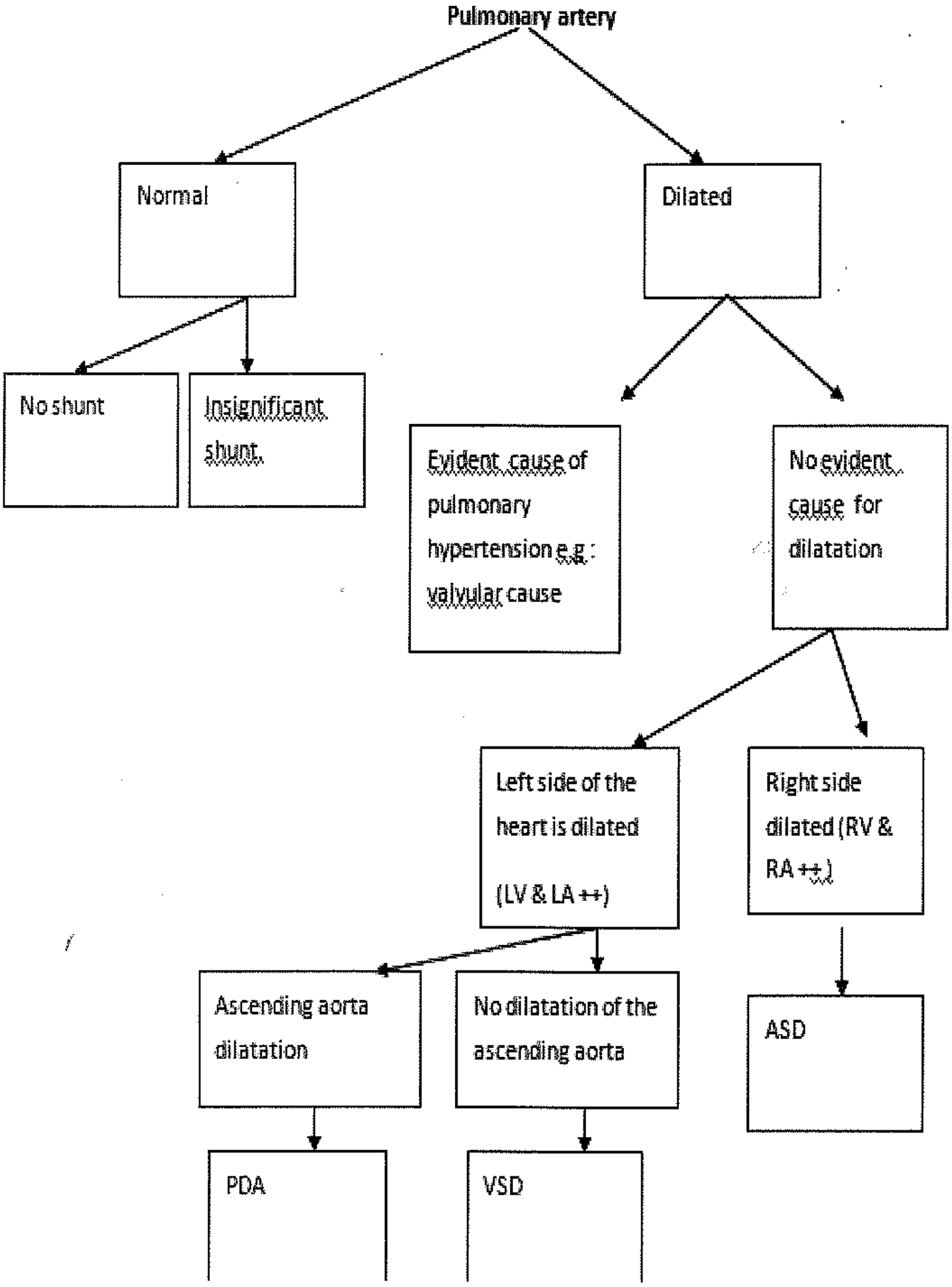
pulmonary < Artery
later → HTN
+ Rt side

N.B : PAPVR will lead to the same heamodynamic effects of an ASD.

EMSE echocardiography algorithm to suspect shunt lesions

This algorithm should never substitute the routine examination of the intracardiac septa by color Doppler,

Rather this algorithm draws the attention for a hidden shunt


Pulmonary artery
Normal
Dilated
No shunt
Insignificant shunt.
Evident cause of pulmonary hypertension e.g : valvular cause
No evident cause for dilatation
Left side of the heart is dilated
(LV & LA ++)
Right side dilated (RV & RA ++)
Ascending aorta dilatation
No dilatation of the ascending aorta
ASD
PDA
VSD

Interventricular septum (IVS) :

2D echo :

The aim by 2 D echo is to

A) measure the IVS dimensions,
B) asses the kinesis of the IVS,
C) to suspect any defects.

A) the end diastolic and end systolic dimensions of the IVS can be measured by 2 D echo "better to be ECG gated" . See diagram 111.

N.B Decreased systolic thickening is a sign of hypokinesis.

B) The kinesis of the IVS should be assessed by 2 D echo and correlated to the M.mode findings, and reported as normal motion, hypokinetic, akinetic, dyskintetic or paradoxical motion.

Wall motion score index can be calculated "used in special situations" :

This is a global score that represents the overall left ventricular wall motion. The aim of this index is the quantitation of wall motion. This methodology involves describing the wall motion characteristics of each of the 16 segments as being either normal (scored "1"), hypokinetic (scored "2"), akinetic (scored "3"), dyskinetic (scored "4") , or aneurismal (scored "5"). A numerical score, typically 1 to 5, is then applied to each of these segments, and the total score is divided by the number of segments evaluated to create a wall motion score index. A ventricle with completely normal wall motion would have a score index of 1.0 (each segment of the 16 segment will be normally scored "1", so the normal score will be 16 / 16). higher scores represents progressively greater degrees of ventricular dysfunction.

C)The sonographer should also search for any defects in the IVS for a more detailed study by Doppler.

M.mode:

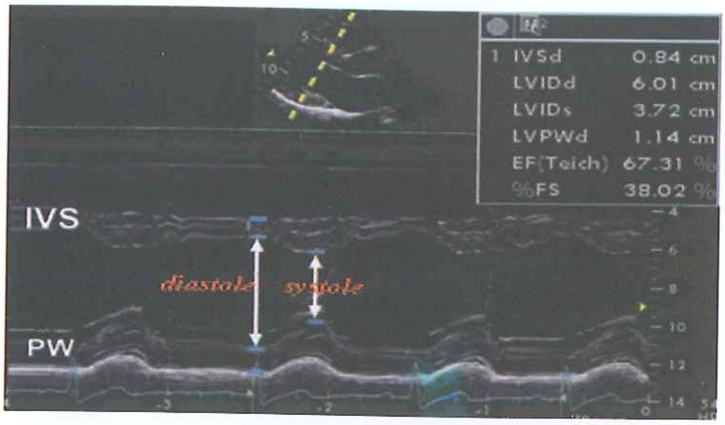
Normal M.mode trace of the IVS : see diagram 143

Hypokinetic IVS in the M.mode : see diagram 144.

Akintetic IVS: see diagram 145.

Paradoxical motion : see diagram 146 A & B.

Dyskinetic motion : see diagram 147.



1 IVSd 0.84 cm
LVIDd 6.01 cm
LVIDs 3.72 cm
LVPWd 1.14 cm
EF(Teich) 67.31 %
%FS 38.02 %

Diagram 143 : Notice the normal motion of the IVS when compared to the posterior wall.

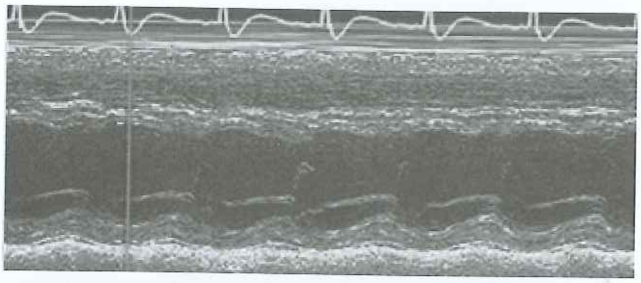


Diagram 144 : Notice the hypokinetic septum when compared to the posterior wall.

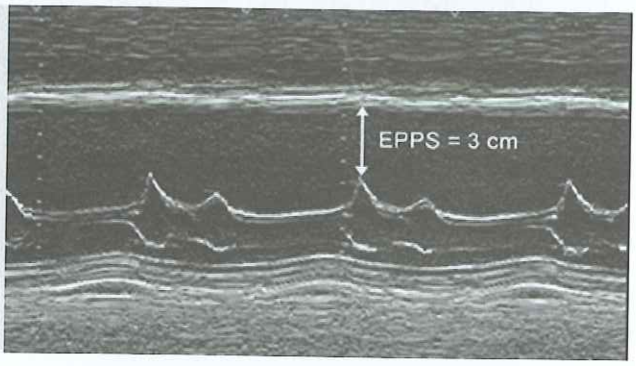
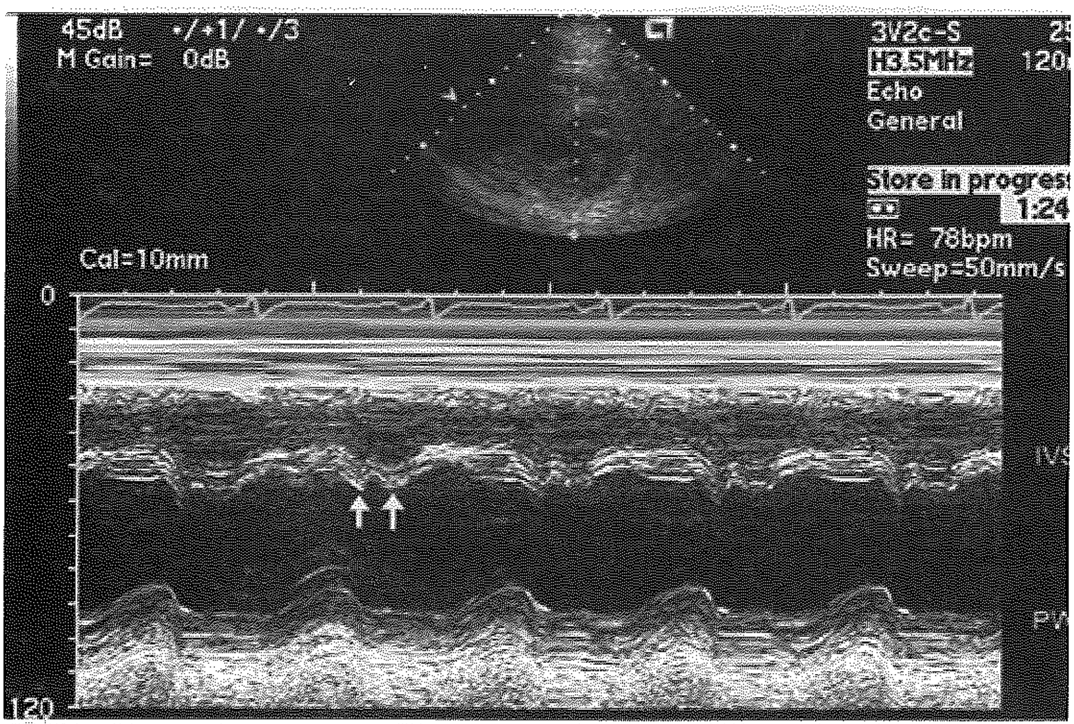


Diagram 145 : Notice the akinetic septum when compaired to the posterior wall.

Diagram 146 A

Diagram 146 for paradoxical motion.



146 B

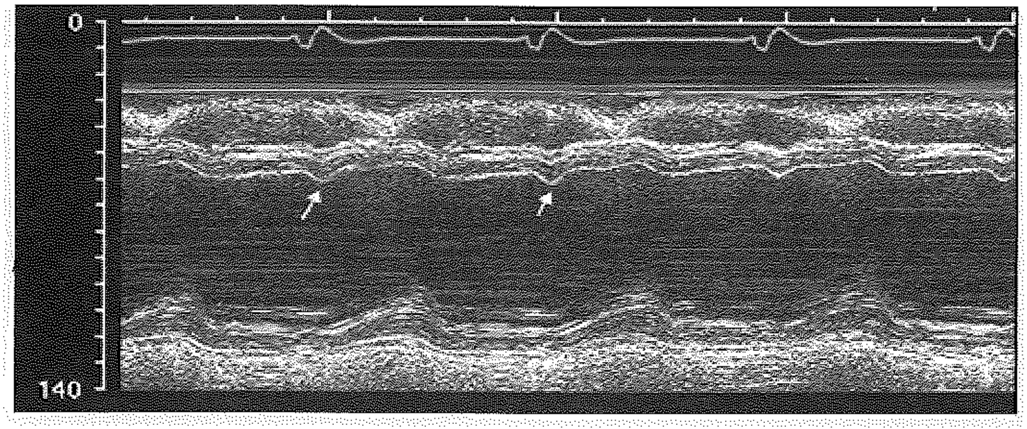
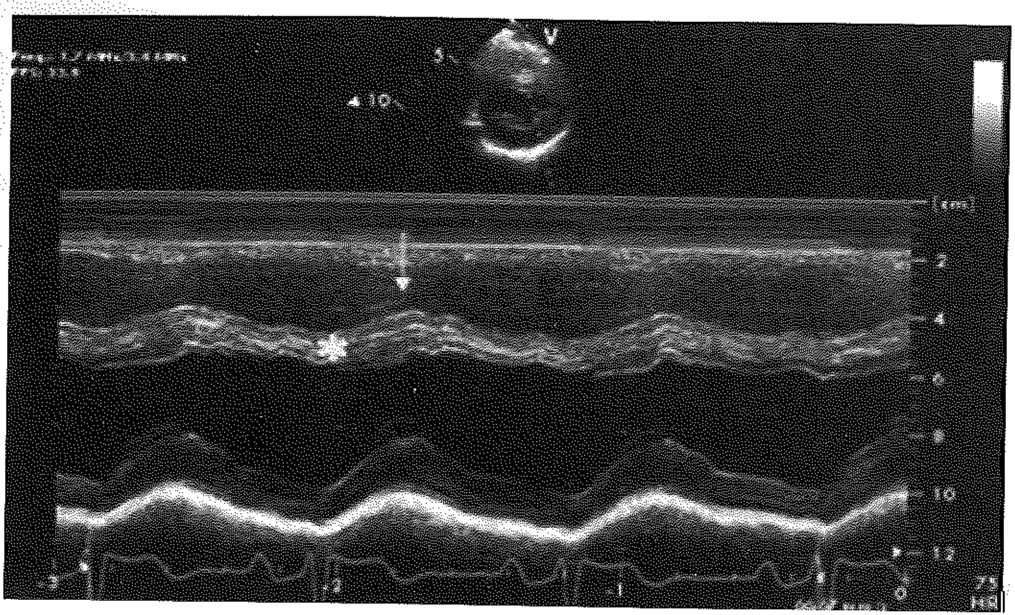


Diagram 147 : showing dyskinetic wall, notice the abnormal septum contractility , notice the absence of IVS thickening.



Doppler :

CW, PW or color Doppler can be aligned in any view showing the IVS. However there is a general consensus in clinical practice to start by the color Doppler.

Typically the spectrum of the color Doppler is optimized to examine the IVS in all parasternal views: the PLAX, PSAX (great vessels level, MV level, Papillary muscle level and apical level). Also it can be used in both of the out flow view and the inflow view, but experience is needed in the latter two views.

The apical views add valuable information too, the IVS should be examined in the apical 4 chamber view and the apical 5 chamber view (and may be the apical 3 chamber view).

N.B * remember to examine the whole IVS. i.e. basal, mid and apical. And in all the mentioned views above.

*Remember that the subcostal views offers a perpendicular cut section over the cardiac septa when using 2 D echo and at the same time a parallel alignment for a Doppler study. See echocardiographic views.

A high velocity jet traversing the IVS indicates a left to right shunt lesion that must be :

1) precisely anatomically localized (upon the sonographer's description the surgeon will choose from various surgical incisions)

2)quantified (Qp/Qs ratio).

Pulmonary stroke volume should normally be equal to the systemic stroke volume.

$$\text{RVOT area (cm}^2\text{) X RVOT VTI (cm)} \quad = \quad \text{LVOT area (cm}^2\text{) X LVOT VTI (cm)}$$

See diagrams 103 & 118.

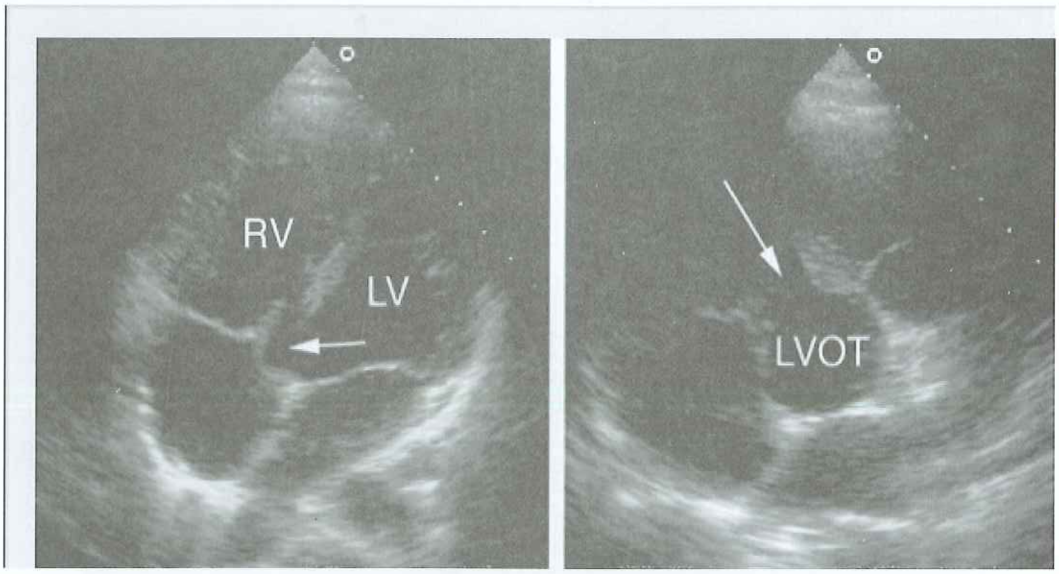
In cases of shunt lesions the volume of blood traversing the RVOT will increase (except with PDA). So the volume of blood traversing the RVOT will be more than the volume of blood traversing the LVOT.

A Qp/Qs more than 1.5 defines a heamodynamically significant shunt that will require intervention.

Localization of the VSD [Types of VSD]:

Before reading the types of VSD, you had better recall from " echocardiographic views" the anatomy of the IVS.

A defect in the inlet septum is called an inlet VSD. This defect is most clearly shown in an optimized apical 4 chamber view between the mitral valve and the tricuspid valve, and at the precise location in the IVS as shown in the PSAX view. See diagram 148. This defect will neither be present in the apical 5ch view nor in the PLAX view. . The surgical incision to close this defect will be in the RA.



Inlet
Apical 4
PSAX

Diagram 148 : Showing an inlet VSD in PLAX and PSAX. The defect is shown by an arrow.

A defect in the perimembranous septum will be called a perimembranous VSD. This defect will be clear in the PLAX view and apical 5 ch view. Its precise location in the IVS will be evident in the PSAX view great vessels level at the perimembranous region as shown in diagram 149. This defect is not seen in the apical 4ch view. The surgical incision will be also in the RA.

Perimembranous
PLAX
apical 5
PSAX

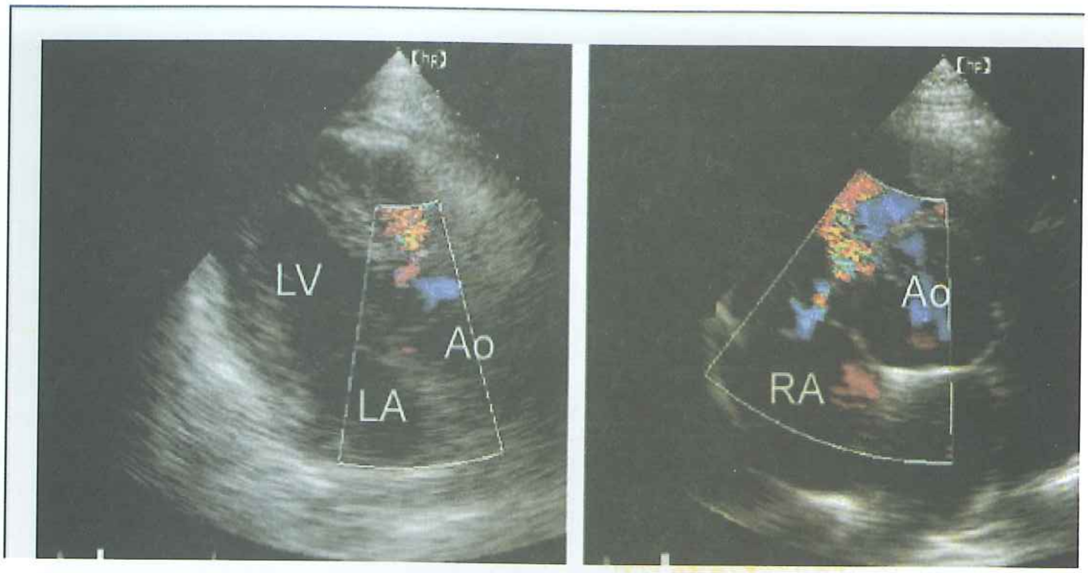
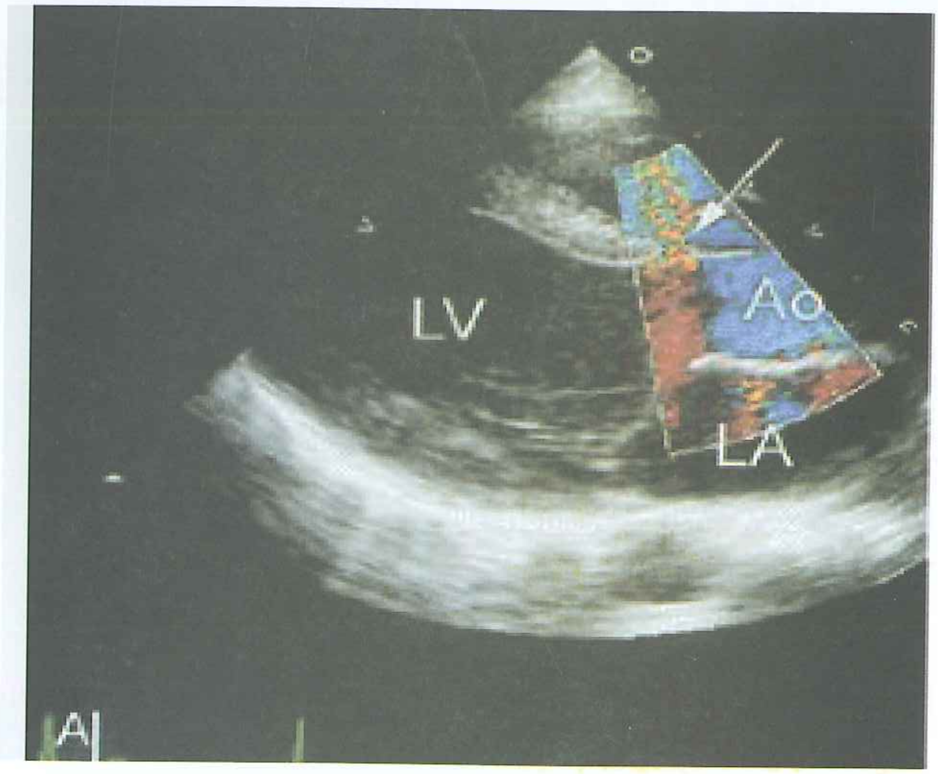


Diagram 149 : showing a peri-membranous VSD, shown in the PLAX and in the PSAX view.

Notice the proximity of the defect to the tricuspid valve. Unlike an inlet VSD, the peri-membranous VSD will be seen in the PLAX and the apical 5 ch views. The inlet VSD will not be shown in the PLAX view.

A defect in the outlet septum will be called an outlet VSD. This defect is also clear in the PLAX view and apical 5 ch view. It's precise location in the IVS will be evident in the PSAX view great vessels level at the outlet region as shown in diagram 150. The surgical incision to close this defect will be in the main pulmonary artery or the ascending aorta.



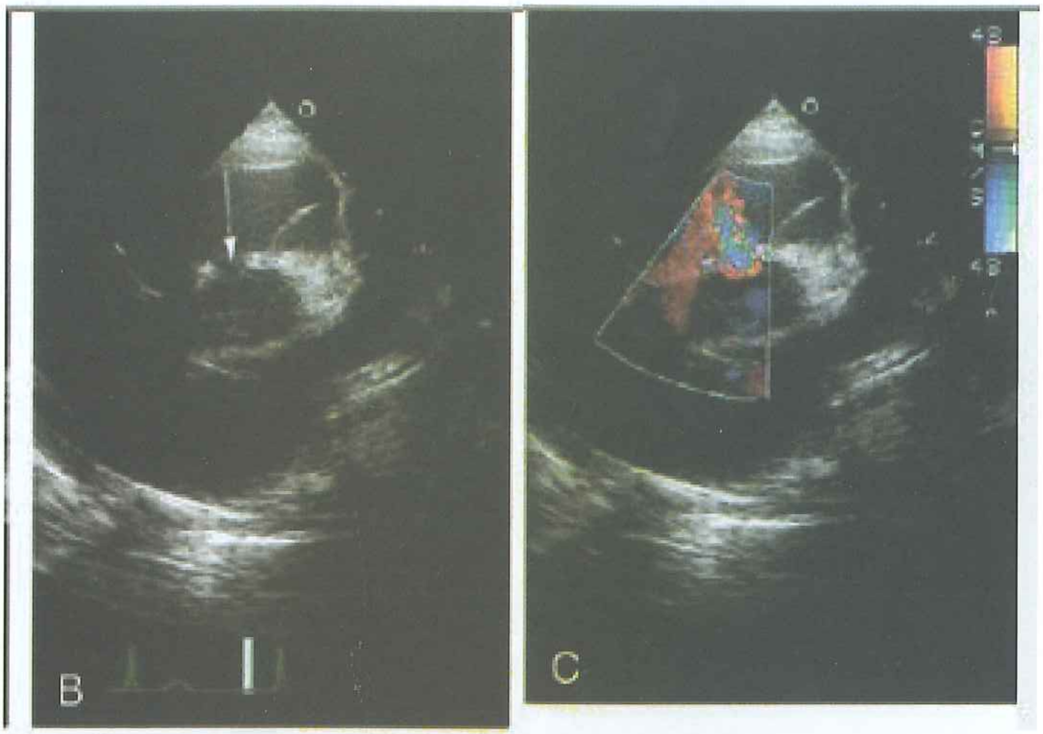


Diagram 150 : showing an outlet VSD, A) PLAX , B & C in the PSAX, notice the precise location of the outlet VSD.

A defect at the IVS just downwards to the pulmonary valve will be called subpulmonary VSD, it will be evident in the PSAX great vessels level near the pulmonary valve. See diagram 151. The surgical incision will be in the main pulmonary artery.

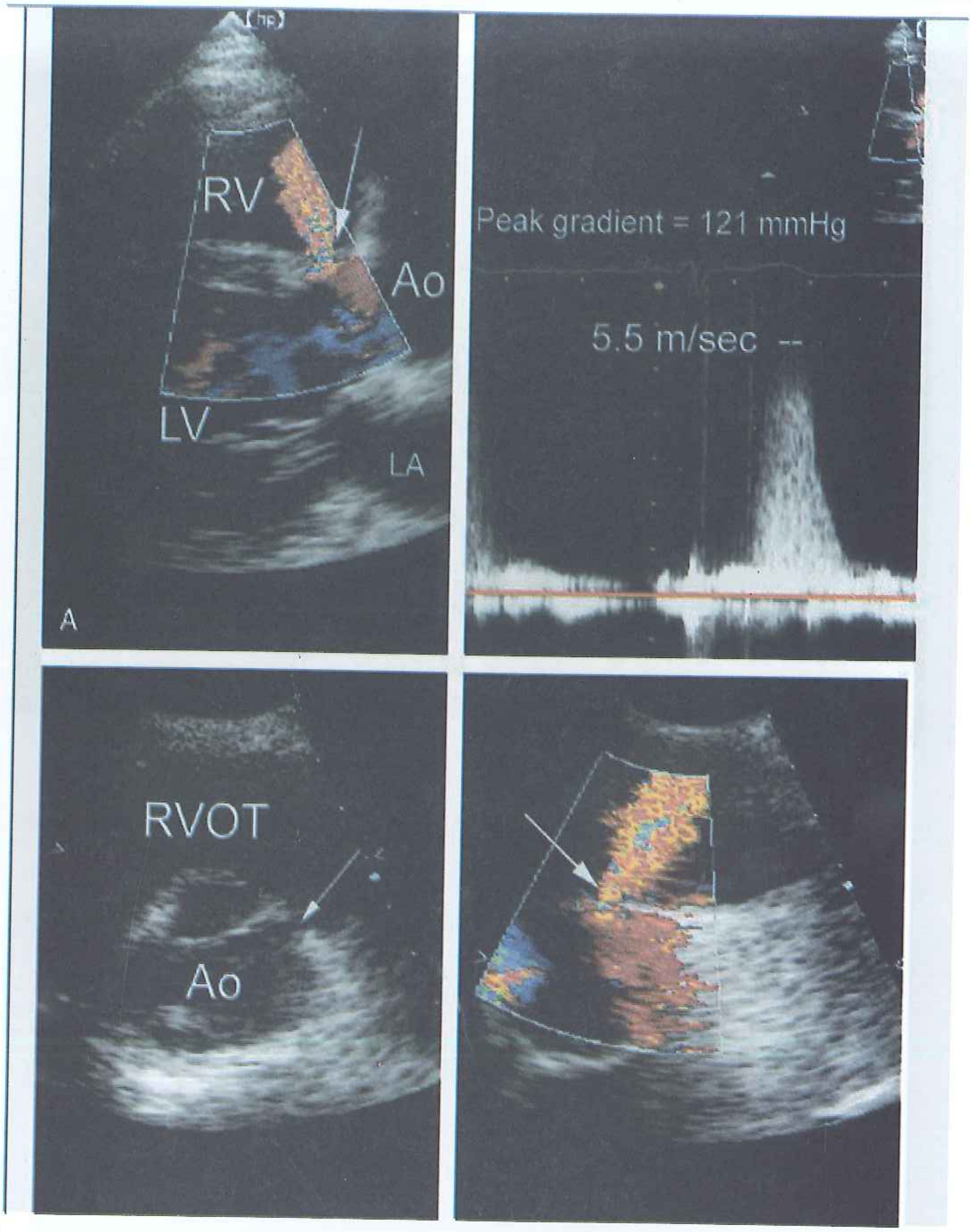


Diagram 151 : sub-pulmonic VSD, notice the precise location just near (below) the pulmonary valve.

N.B sometimes the VSD's have extensions, e.g. subpulmonic VSD with outlet extension or perimembranous with inlet extension. The detailed description of these defects is beyond the scope of this book.

A faulty description will lead to a faulty surgical incision, so experience in a tertiary center intervening in such cases is needed before self confidently taking these decisions.

Contrast echocardiography is so helpful in some clinical situations, but it is not available in Egypt.

Inter atrial septum (IAS):

2D echo :

The aim of the 2 D echo is to suspect any abnormality in the IAS. These mainly include masses (see "intra cardiac masses), IAS aneurysm and defects.

There is a general consensus that the echocardiographer should not judge the presence of an ASD using only 2 D echo especially from an apical 4 chamber view, because part of the IAS may look dropped out giving a false impression of an ASD. And if an ASD is suspected it must be confirmed by:

1) Color Doppler.

2) Presence of the specific hemodynamic effects.

An IAS aneurysm is a redundant part of the IAS that moves from side to side and crosses more than 1 cm beyond the midline. See diagram 152.

These IAS aneurysms are sometimes associated with a PFO or an ASD, that can be confirmed by color Doppler or contrast echo. Sometimes a TEE study is indicated especially if the patient suffered a previous stroke, otherwise the **EMSE** recommends that as long as there is neither a hemodynamic effect from the suspected PFO/small ASD complicating the IAS, nor the patient suffered a previous stroke or is at increased risk to suffer paradoxical emboli (A.F, Left atrial appendage, performing a diving sports, etc), no further investigations seem rational.

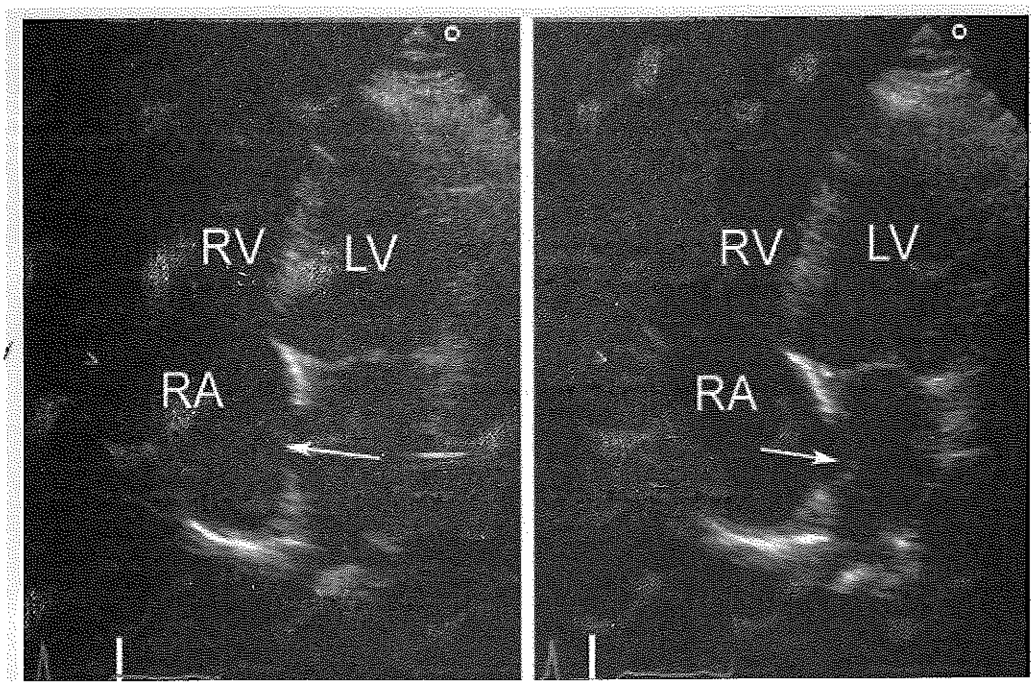


Diagram 152 : An interatrial septal aneurysm shown by the arrow moving from side to side.

M.mode:

The M.mode adds little information to the assessment of the IAS. Anatomical M.mode (M.mode steer) can be used if seemed applicable.

Doppler :

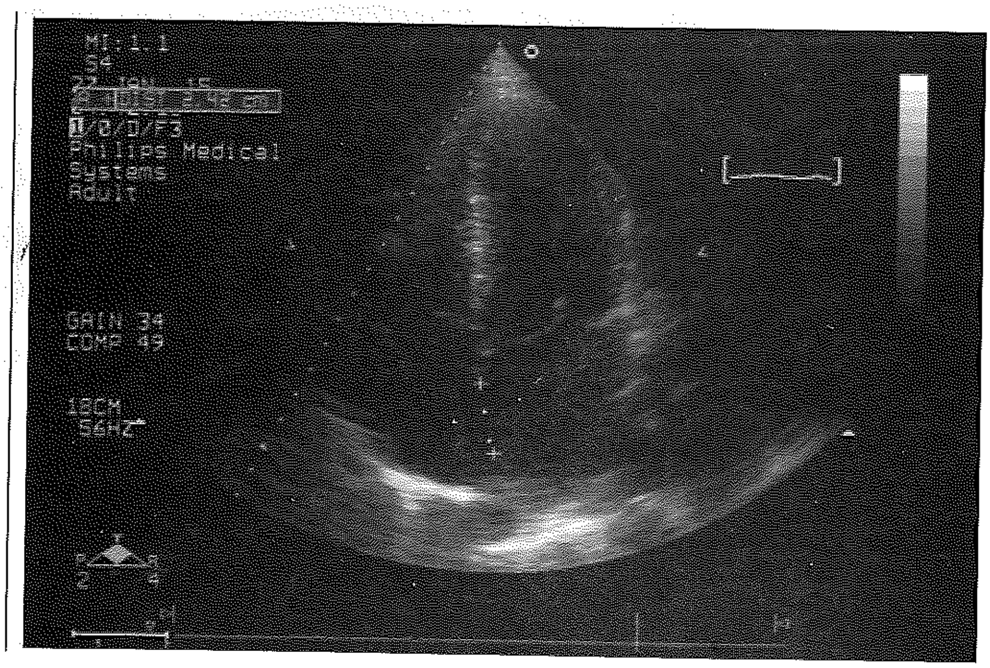
There is a general consensus that color Doppler is the preferred modality to examine the IAS specially when looking for defects.

An ASD must be searched for in all views showing the IAS. Typically the subcostal views offers better imaging for the ASD in most cases.

Before adjusting the color Doppler on a suspected ASD, the sonographer should decrease the Nyquist limit (color PRF) to a lower value i.e. 40 cm/s in order to be able to predict the low velocity jets with a better mosaic pattern.

A mosaic pattern traversing the IAS carries very high sensitivity and specificity for the diagnosis of an ASD. However dropping outs in the IAS may complicate the picture giving a false impression by color Doppler that there is a left to right flow specially when interpreted by a beginner sonographer. Diagram 153 is an example of a dropping out in the IAS. To overcome this pitfall a beginner echocardiographer should first look for the effect of the ASD, reexamine the IAS in the subcostal views.

diagram 153 : intact IAS but showing a dropped out part, this dropping out may be Misinterpreted by a begginer echocardiographer as an actual defect, notice the normal sized RV



false measurement of a dropped out IAS for an ASD.

Types of ASD :

Before reading the types of ASD it is better to recall the anatomy of the IAS from "the echocardiographic views" .

1) A defect in the septum premium is called a primum type ASD. This type is confirmed by the characteristic position of the mitral and the tricuspid valves as arising side to side with the loss of the normal apical displacement of the TV. See diagram 154.
   Best examined in subcostal and apical 4 chamber views.

Also the absence of a rim between the ASD and the atrio-ventricular valves confirms an ostium premium ASD. See diagram.

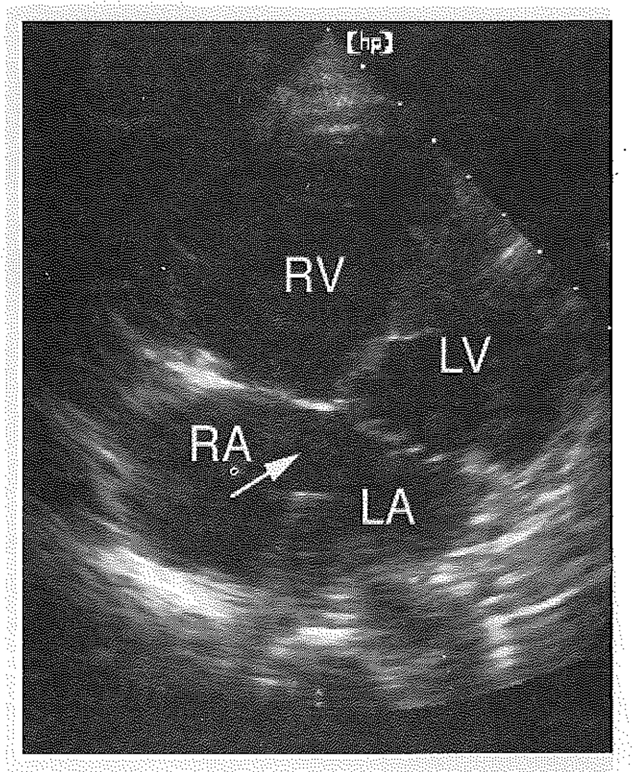


Diagram 154 : Showing ASD premium , see text for details.

2) A defect at the site of the septum secundum is called a secondum ASD. There must be a rim between the defect and the atrio-ventricular valves (even if it is a small rim it will still be considered as an ASD secundum). In this type the normal apical displacement of the TV is preserved. See diagram 155. Best examined in subcostal and apical 4 chamber views.

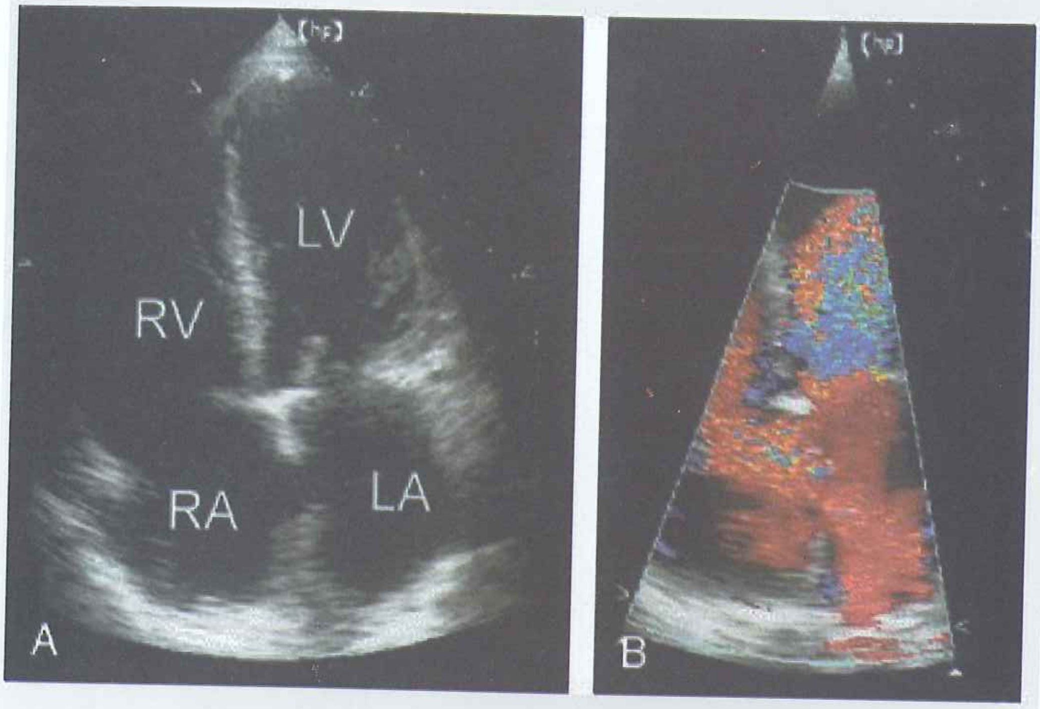
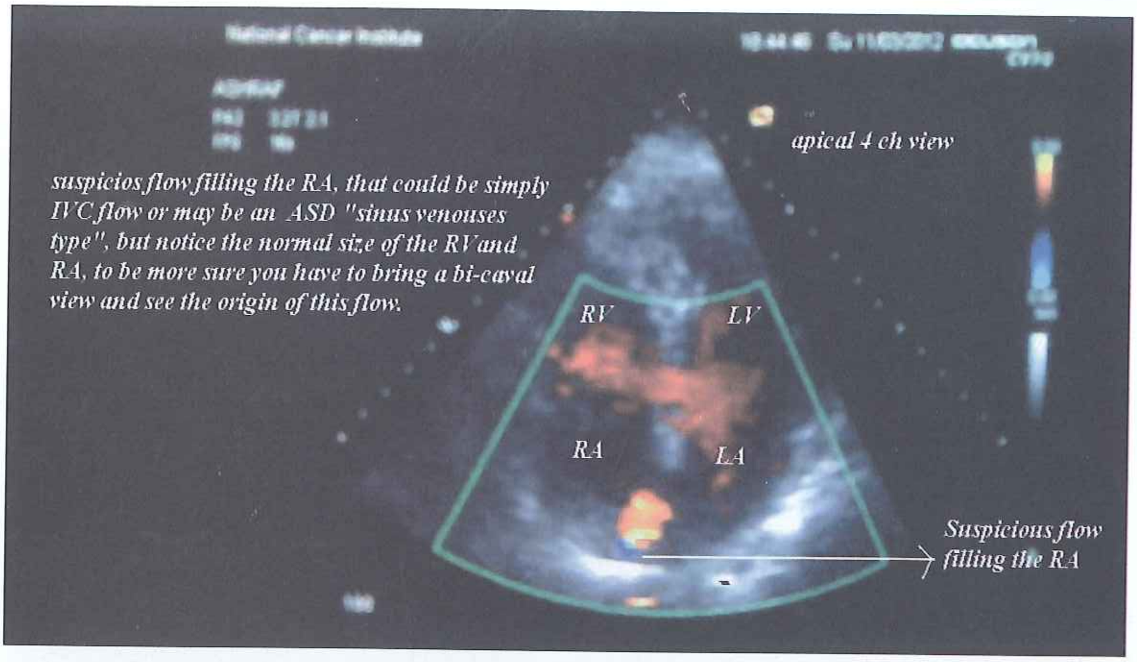


Diagram 155 : Showing secundum ASD, notice the flow from the LA to the RA, see text for details.

3) A defect in the sinus venosus IAS is called a sinus venouses ASD. Recall from "the echocardiographic views" the region of the fusion between the smooth and the rough parts of the IAS. This defect is characterized by its proximity to the SVC (very rare the IVC)
Best examined in the bicaval view.
N.B : exaggerated flow from the SVC may raise false suspicion for an ASD sinus venosus defect in an apical 4 chamber view. See diagram.
Exclusion of a siunus venosus ASD will be made on basis of the hemodynamic effects and from a bicaval view demonstrating the origin of the flow. See diagram 156 A & B



suspicios flow filling the RA, that could be simply IVC flow or may be an ASD "sinus venouses type", but notice the normal size of the RV and RA, to be more sure you have to bring a bi-caval view and see the origin of this flow.

Diagram 156 A : Showing a suspected jet flow filling the RA. To be confirmed from a subcostal bi-caval view. As below in diagram 156 B.

N.B notice the color artifact traversing the IVS, this may confuse a beginner echocardiographer especially when interpreted in a frozen view. (For beginners simply revise the description of the high velocity jet discussed before.)

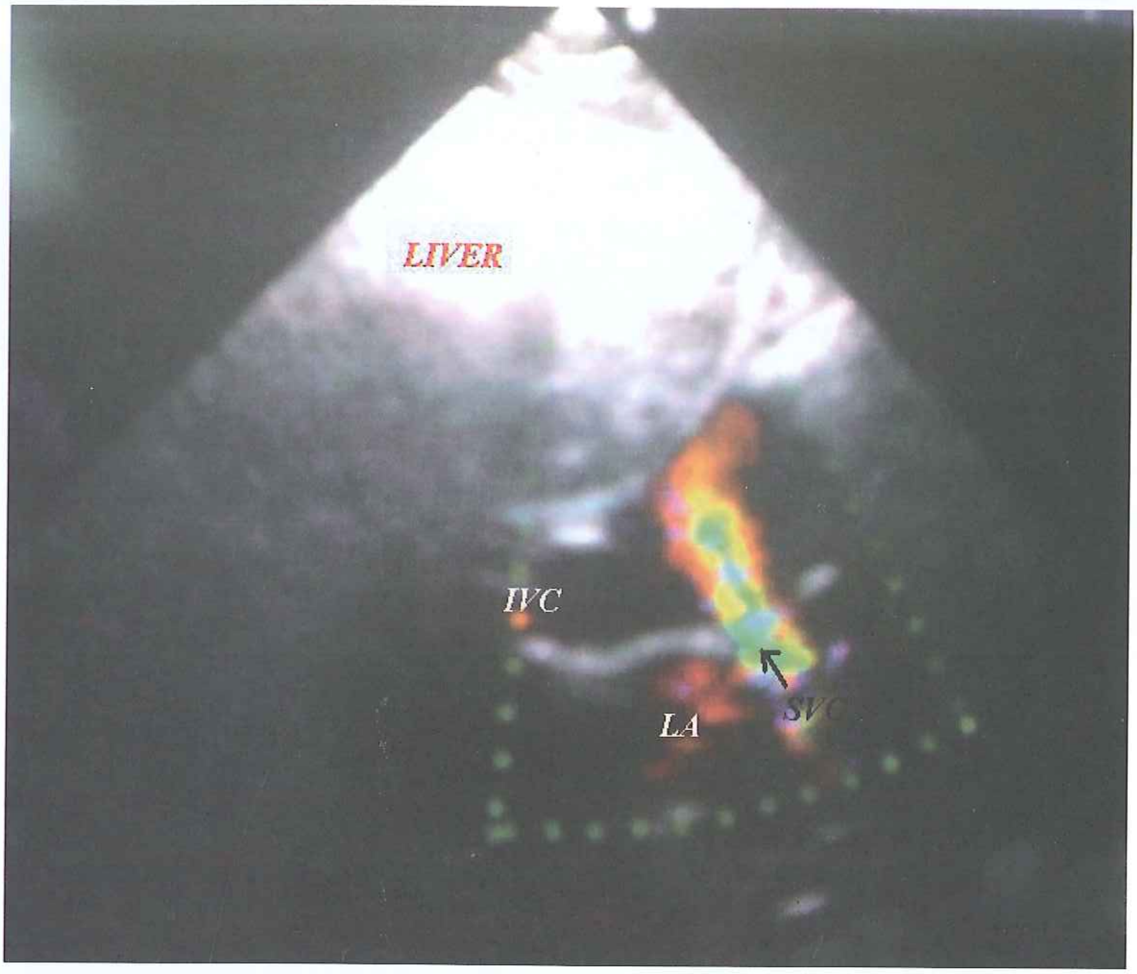


Diagram 156 B : The same patient in 156 A, confirming that the direction of the flow is from the SVC into the RA (denoted by the arrow) thus excluding the sinus venouses ASD.

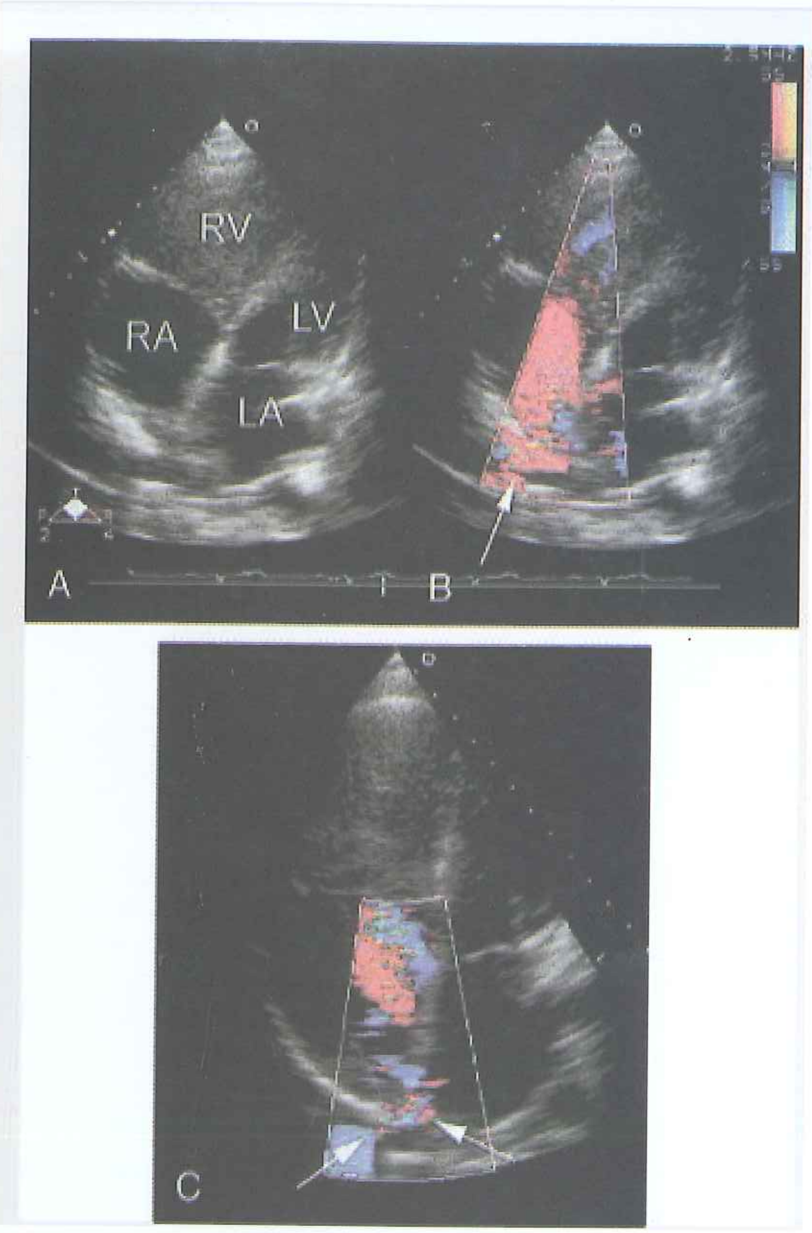


Diagram 157: An actual ASD sinus venosus type, notice the similarity between this flow and the normal SVC flow. However the dilated RV suggests that there is a left to right shunt. In this situation the flow will arise from the LA to the RA in the bi-caval view.

4) Coronary sinus type ASD :
   In this situation there is no actual defect in the IAS, rather there is deroofing of the coronary sinus into the left atrium (i.e. the left atrium opens "abnormally "in the coronary sinus). So, the blood will flow from the higher pressure LA to the lower pressure coronary sinus that eventually drains into the RA. The end result is that the blood is shunted from the left side to the right side resulting in the same hemodynamic effect of an ASD.

   This defect is diagnosed by suspicion, a dilated coronary sinus must draw the attention for this anomaly. A high velocity jet detected by color Doppler filling the coronary sinus plus the hemodynamic effects of the ASD gives clues for diagnosis.

   Contrast echo plays a critical role in this situation.

Patent ductus arteriosus (PDA):

The PDA can be examined in the modified view "high parasternal short axis view great vessels level" i.e. Ductal level view.

2D echo :

The PDA can be measured as shown in the diagram 158.

The hemodynamic effects of the PDA can be assessed by 2 D echo in terms of LV, LA and ascending aortic dilatation.

M.mode:

Adds little information for the diagnosis of PDA, and can be used to asses various dimensions as in the 2 D imaging.

Doppler :

Color Doppler is the main modality for the diagnosis of the PDA, in a ductal level view color Doppler can be adjusted upon the pulmonary bifurcation with the descending aorta in between. See diagram 158.

A high velocity mosaic jet flowing from the descending aorta to the left pulmonary artery plus the hemodynamic effects of the shunt diagnose the condition. See diagram 160.

The shunt can be measured using the color Doppler but take into account that you have to adjust the color gain an d the Nyquist limit, as an increase in the color gain or a decrease in the Nyqiust limit will lead to an over estimation of the orifice of the PDA.

CW Doppler : a positive continuous Doppler signal can be seen as shown in the diagram 159.

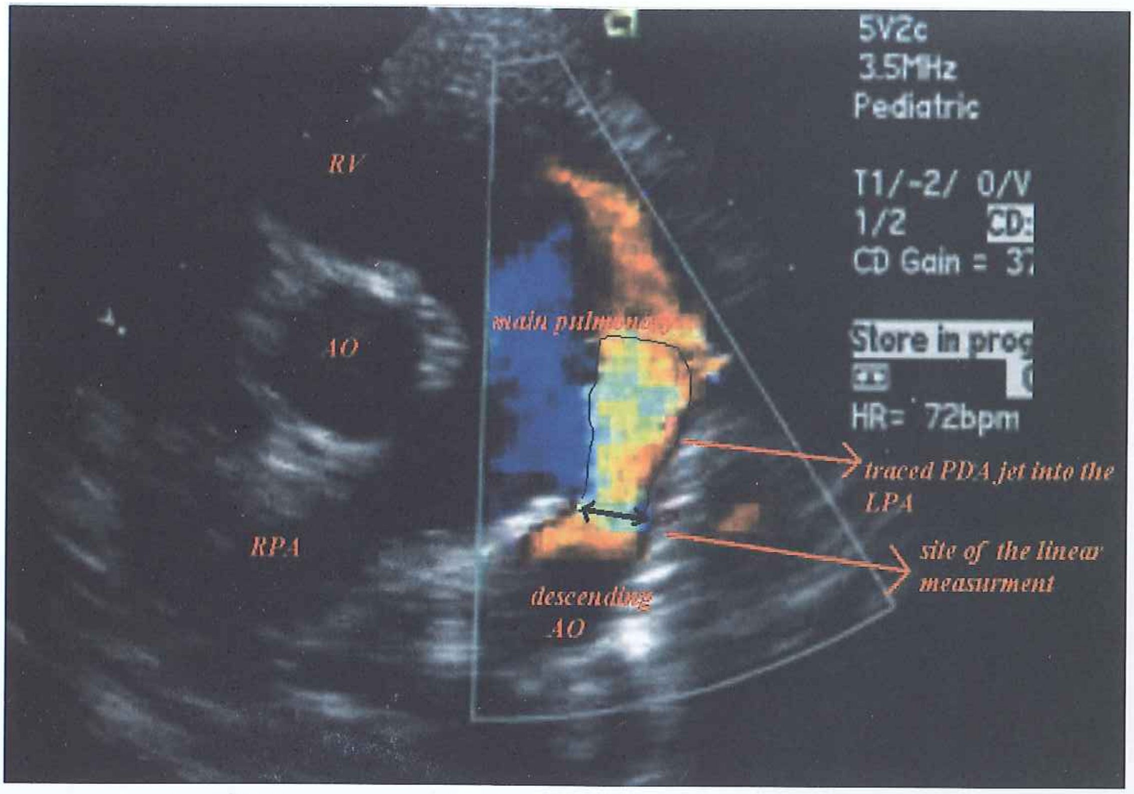


Diagram 158 : Showing PDA jet in high PSAX view (ductal level view) and the site of linear measurement of size of the PDA. (to bring the ductal level view, simply slide upwards from the great vessels level position until the LA becomes replaced by the RPA.

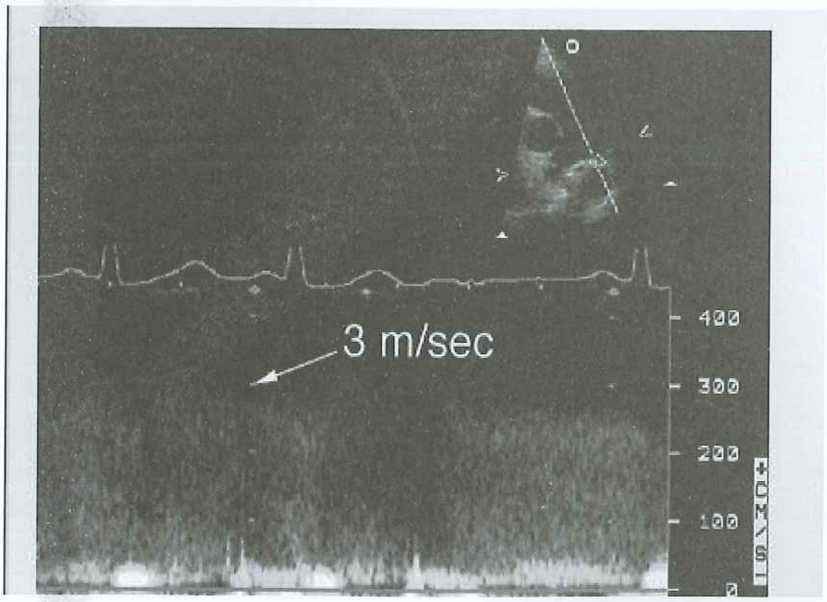


Diagram : 159 shwoing the high velocity signal elicited by the CW doppler. Notice the Continous flow.

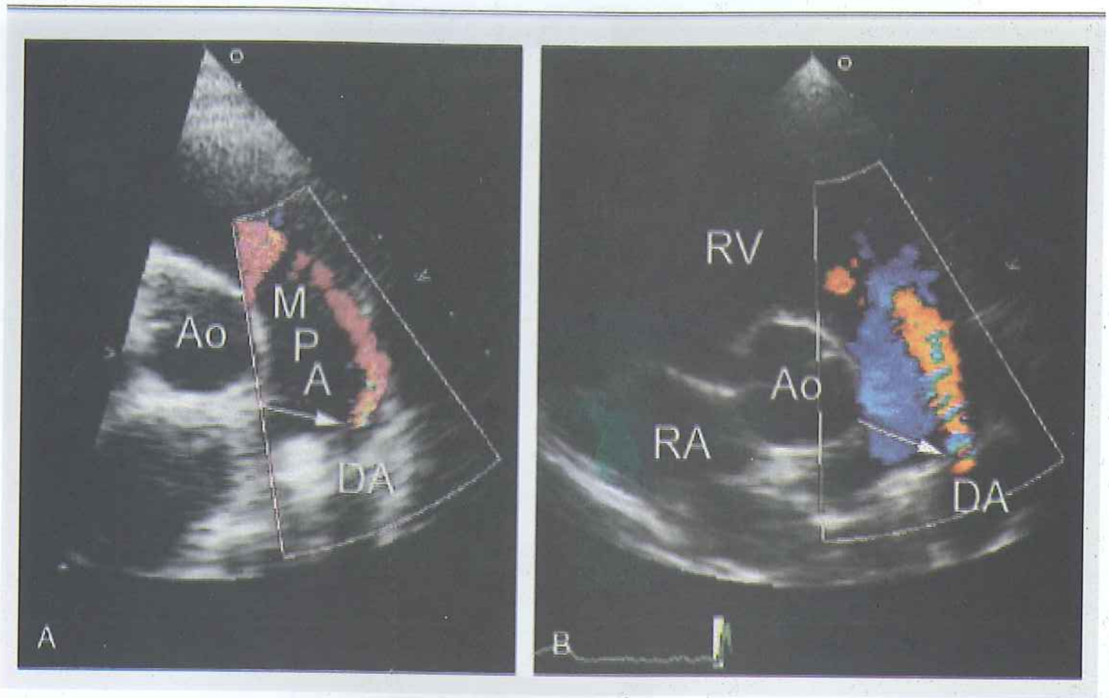


Diagram 160 : showing PDA by color Doppler in PSAX great vessels view.

PAPVR:

2D echo :

In this condition one or more of the pulmonary veins anomalously openes into the RA or one of its tributaries instead of opening into the LA.

This anomaly needs a high level of suspicion; remember that the “effect of the lesion is more important than the defect”; the hemodynamic effects is the same as in ASD.

The first clue is a dilated right side of the heart or a dilated coronary sinus in the absence of a clear cause.

A clear suprasternal short axis view (SSSA view) will be diagnostic for the condition. Unfortunately it is usually difficult to acquire in adult patients.

If the SSSA view is clearly aqiured, the absence of one or more of the pulmonary veins is confirmatory. If this view was not optimized, contrast echo will be very helpful to detect the level at which the shunt is present.

M.mode: adds little information to the diagnosis.

Doppler :

Color Doppler is the main modality to search for the flow of the pulmonary veins, either if the four pulmonary veins drains into the LA in the SSSA view or one pulmonary vien is missing.

The precise localization of the drainage of the PAPVR is beyond the scope of this book but here are some clues for it:

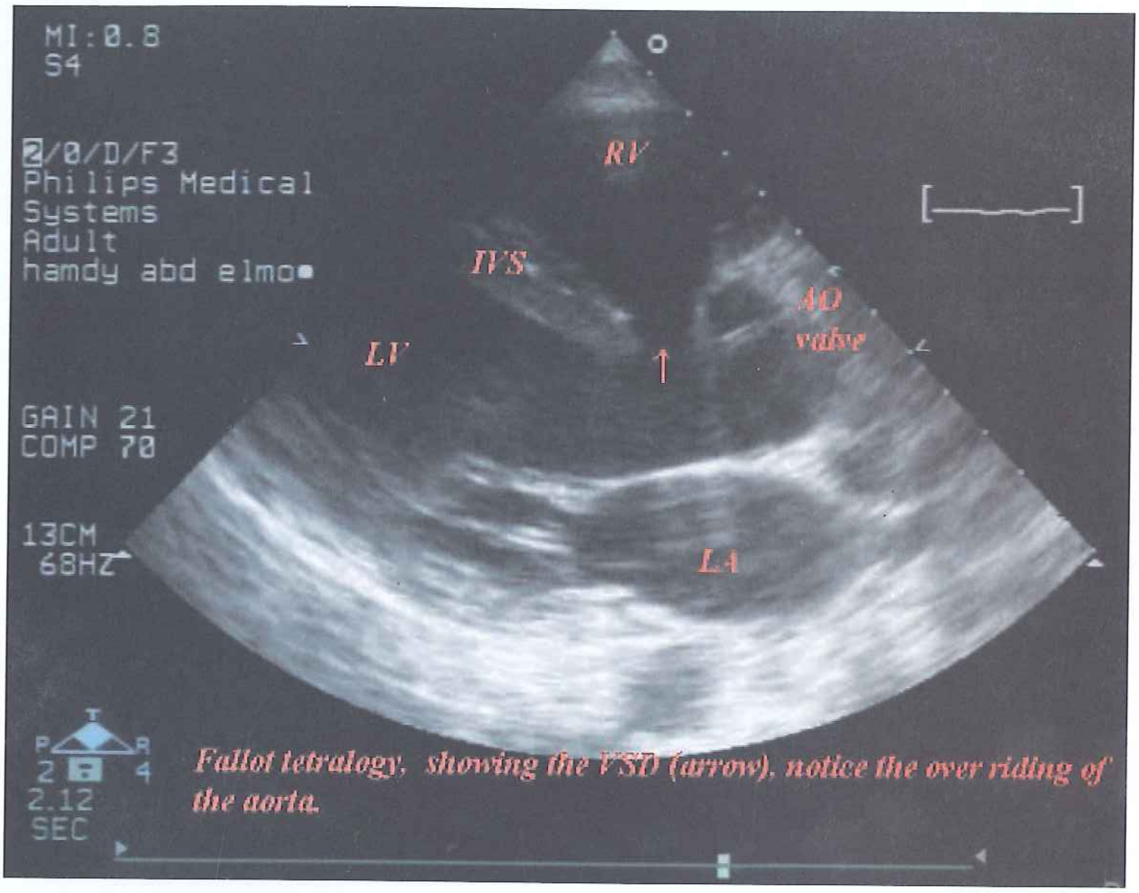
The color Doppler can be used to search for the flow of the anomalous pulmonary vein as it drains into the right atrium or into the right atrial tributaries, i.e. where does it drain?. The sonographer can search for its flow in the SVC (SSSA view), IVC (bicaval view) coronary sinus (long axis coronary sinus view) or left inominate vein (SSSA view)

Fallot tetralogy :

Fallot tetralogy includes a group of abnormalities. The most two important anomalies are the VSD (as large as the aortic annuals) and the pulmonary stenosis (PS) (see diagram 161 A & B). The VSD is usually due to mal- alignment of the intra cardiac septation leading to overriding of the aorta. Right ventricular hypertrophy is secondary to pulmonary stenosis. In the classic form of fallot, as the blood encounters RVOT obstruction (PS), the blood will shunt through the VSD into the relatively lower pressure chamber, namely the LV, leading to cyanosis. For sure that some volume of blood flows through the PS. According to the severity of the PS and the size of VSD, the degree of cyanosis will be determined, and also. In fallot tetralogy the shunt is almost always bidirectional.

An emergency situation called cyanotic spell may occur in these patients suffering fallot tetralogy. It is due to a decreased systemic vascular resistance (due to any cause e.g. Crying) in which the blood shunting will be exclusively right to left, leading to a severe form of cyanosis and a vicious circle of tachypnea → leading to increased venous return→ with increased right to left shunting and oxygen de-saturation→ that will lead to more tachypnea → and so on.

Treatment of such a "sometimes fatal condition" includes squatting position, I.V morphine, I.V non selective beta blocker, and in some instances the use of a ventilator. (Treatment details are beyond the scope of this book)



Fallot tetralogy, showing the VSD (arrow), notice the over riding of the aorta.

Diagram 161 A: Showing a malalignment VSD. With overriding of the aorta, the blood flows from the RV through the VSD and towards the aorta.

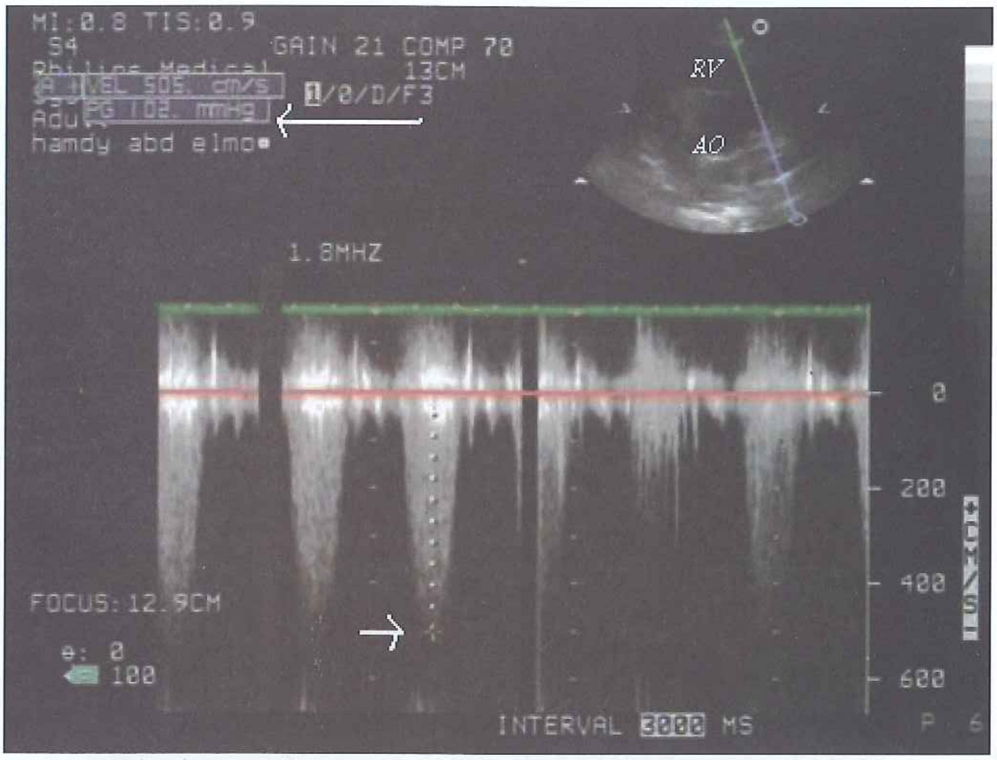


Diagram 161 B: CW Doppler over the RVOT, pulmonary valve and main pulmonary artery in high PSAX view. Notice the high velocity jet denoting severe pulmonary stenosis.

Chapter 15: Assessment of the pericardium :

2D echo : ***the main modality for assessment.***

The pericardium can be seen as the most white outermost layer of the heart, can be seen in any view but best in the PLAX view.

The thickness of the pericardium can be measured but correlates poorly with the actual pathology.

In general the thickness of the pericardium should not exceed 2mm.

N.B MRI will be indicated for more accurate assessment of the pericardium.

Various pathologies that can be seen by 2 D echo :

1) **Thickened pericardium:**
   A thickened pericardium may raise suspicion for a constrictive or restrictive pathologies (see diagram162) that can be confirmed by PW Doppler .

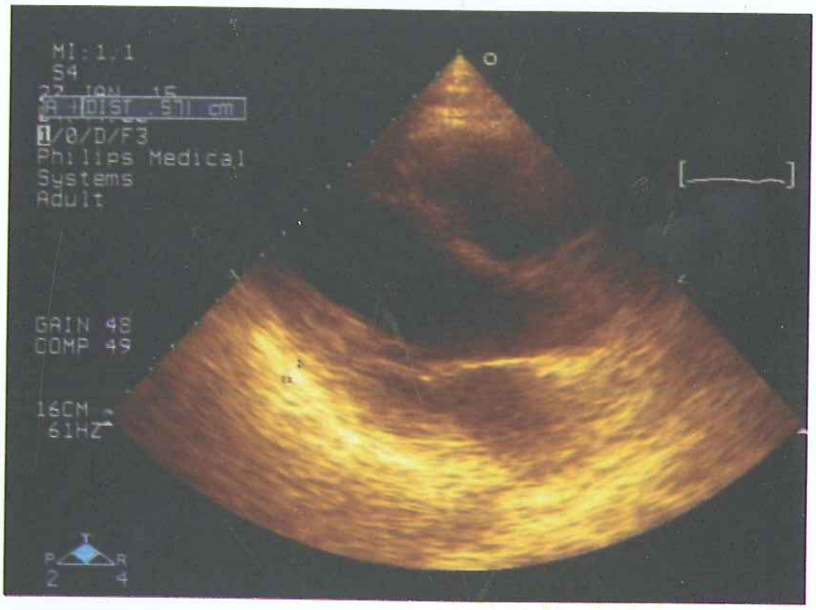


Diagram 162 : Thickened pericardium

2) **Pericardial fluid :**
   A) **Pericardial effusion :**
      2D echo :

   *A fluid accumulating in between the two pericardial layers must be investigated accurately, to be differentiated either pericardial or pleural in origin.
   This will be done using the "descending aorta" as an anatomical land mark.
   Normally, no pericardium covers the descending aorta, so, no pericardial effusion can be seen posterior to the descending aorta, and any fluid posterior to the descending aorta will be mostly

a pleural effusion. And any fluid anterior to the descending aorta will be mostly a pericardial effusion. See diagram 163 A & B.

*Also the presence of the hemodynamic effects of the effusion, in terms of RV collapse or RA collapse confirms the diagnosis of the pericardial effusion.
(Plural effusion does not cause such cardiac effects)

Grading the severity of the effusion :

By 2 D echo the fluid can be measured as shown in the diagram.
See the table below for grading of the severity.

Grading of severity of pericardial effusion by 2 D echo (by measuring the thickness of the echo free space as shown in the diagrams below).

Mild	Moderate	Severe
< 1cm → about 100 ml	1-2 cm → about 400 ml	>2 cm → about 500 ml or more.

Look for any masses, cysts, or fibrin threads seen within the effusion (or at any other site), these findings will direct the treatment plan and the clinical diagnosis.
NB: The fluid is either pus, blood or serous, it will be difficult to confirm by echo.

Also hematomas have the same echocardiographic density of the myocardium and it will be difficult to confirm. Localized effusions are difficult to be seen by TTE, but are almost always suspected in a post cardiac surgery patient with unclear cause of hemodynamic deterioration and unexplained cause of chamber compression, in this case a TEE is indicated.

One of the common sites missed by TTE study and diagnosed by a TEE exam is a localized effusion posterior to the RA.

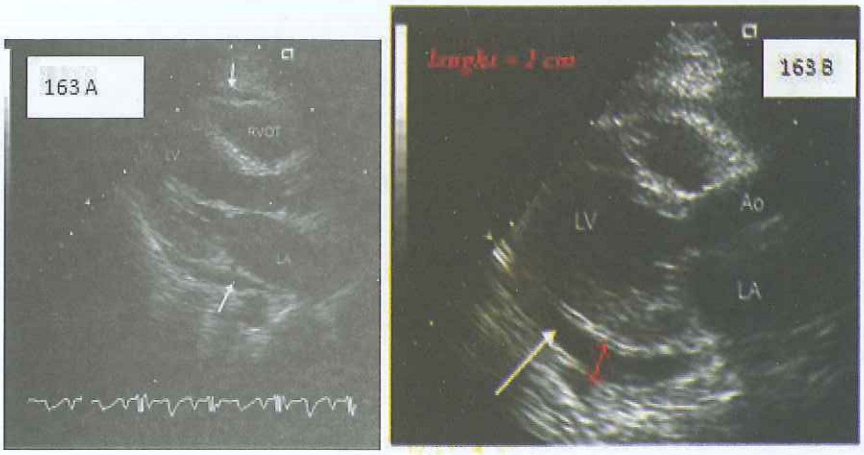


Diagram 163 A & B : showing two different cases of pericardial effusion, notice the echo free space denoted by the arrows, also notice the position of the fluid being anterior to the descending aorta. 163 B showing an example for measuring the pericardial effusion for grading of the severity.

B) Cardiac tamponade :

Cardiac tamponade is a condition where the pericardial pressure has critically increased to the limit that it is compressing the cardiac chambers that will eventually lead to impairment of the cardiac filling with subsequent low cardiac output status.

Tamponade can be classified as a clinical tamponade and echocardiographic tamponade. But the clinical signs are more important than the echocardiographic signs.

Clinical signs simply include any mark of hemodynamic deterioration: Dropping of B.P, faint pulse, cold extremities, sweating, deteriorating dyspnea, orthopnea or oliguria. The decision to intervene with pericardiocentesis must not be delayed.

2 D echo :

The echocardiographic findings of tamponade include the compression of any chamber, but the right ventricular diastolic collapse and the exaggerated right atrial systolic collapse are the most common encountered signs. See diagrams 164 & 165.

Normally the RV fills during the diastole, any compression during this filling phase (diastole) is diagnostic for tamponade and watchful clinical monitoring is mandatory.

Normally the RA collapses during the systole, an exaggeration of this collapse is suggestive of tamponade.

See diagram 166 to 169 showing different cases of pericardial effusion,.

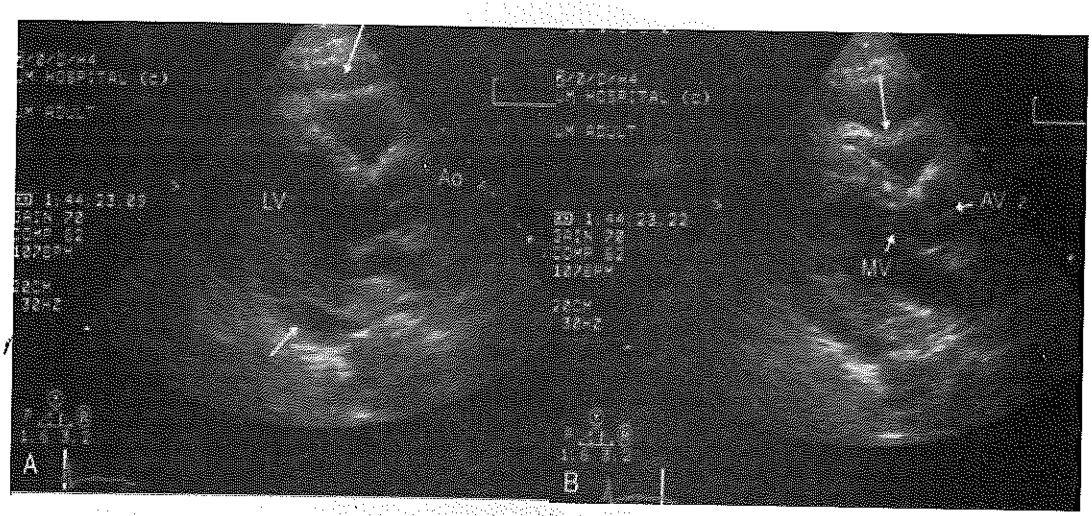


Diagram 164 A & B. A: mild RV collapse.
B : marked RV diastolic collapse.

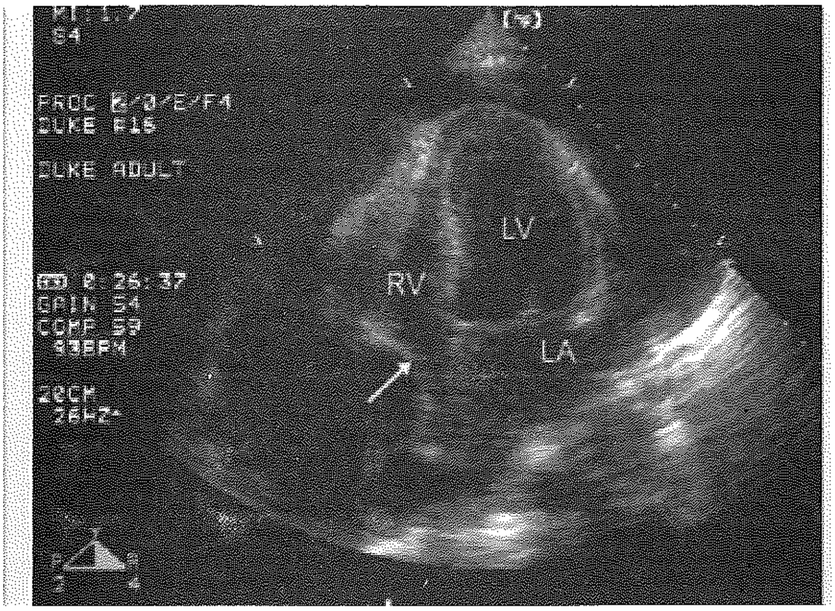


Diagram 165 : showing RA systolic collapse

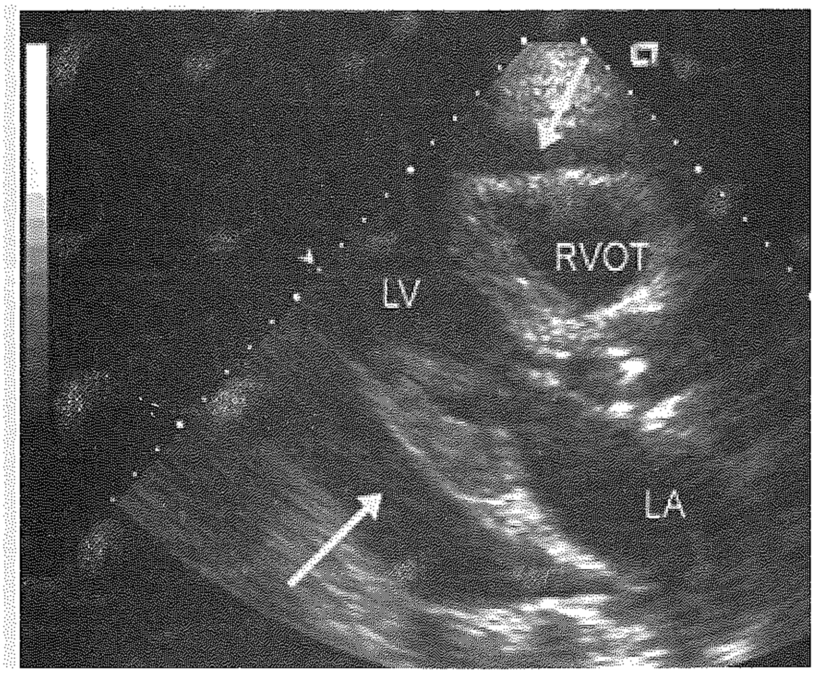


Diagram 166 : showing a severe case of pericardial effusion in PLAX view.

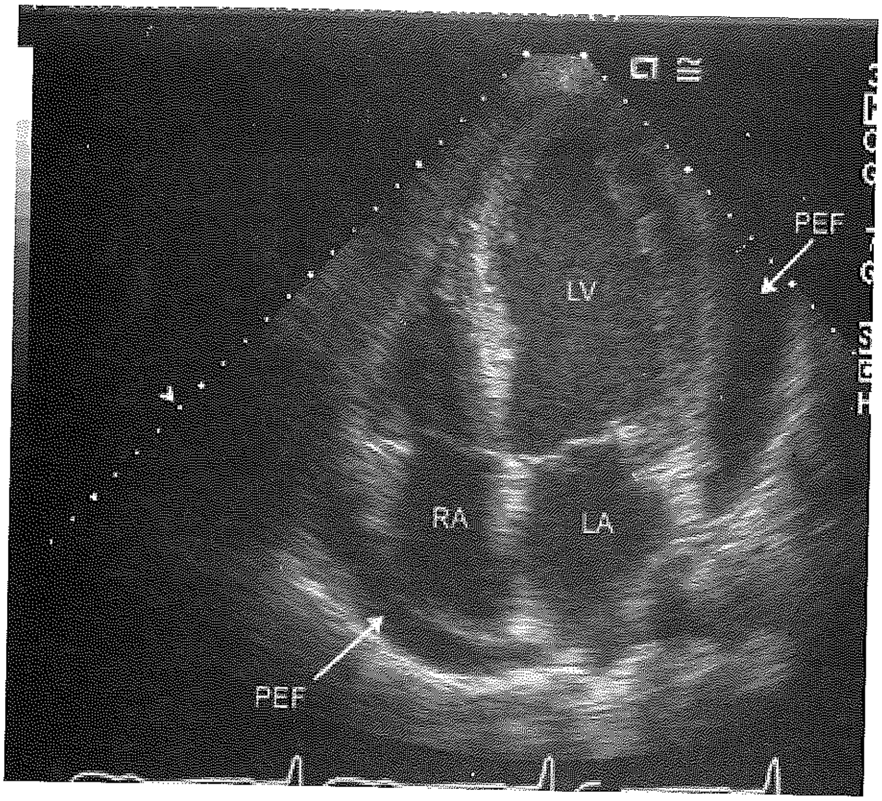


Diagram 167 : moderate case of pericardial effusion in apical 4ch view.

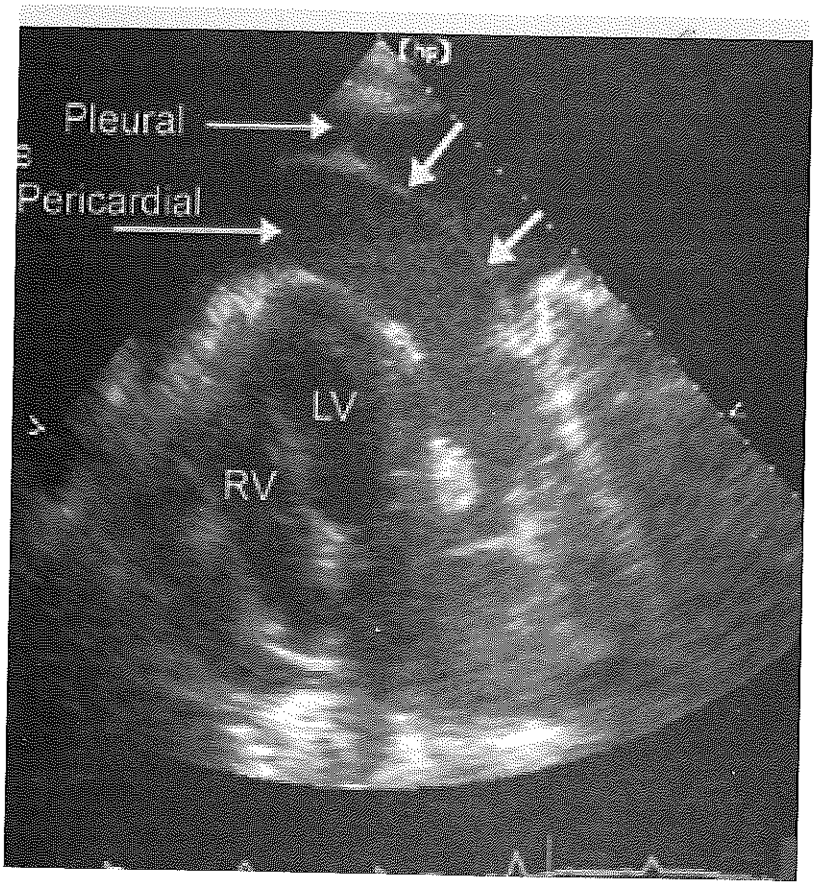


Diagram 168 : combined case of pericardial effusion and pleural effusion in an apical 4 ch view.

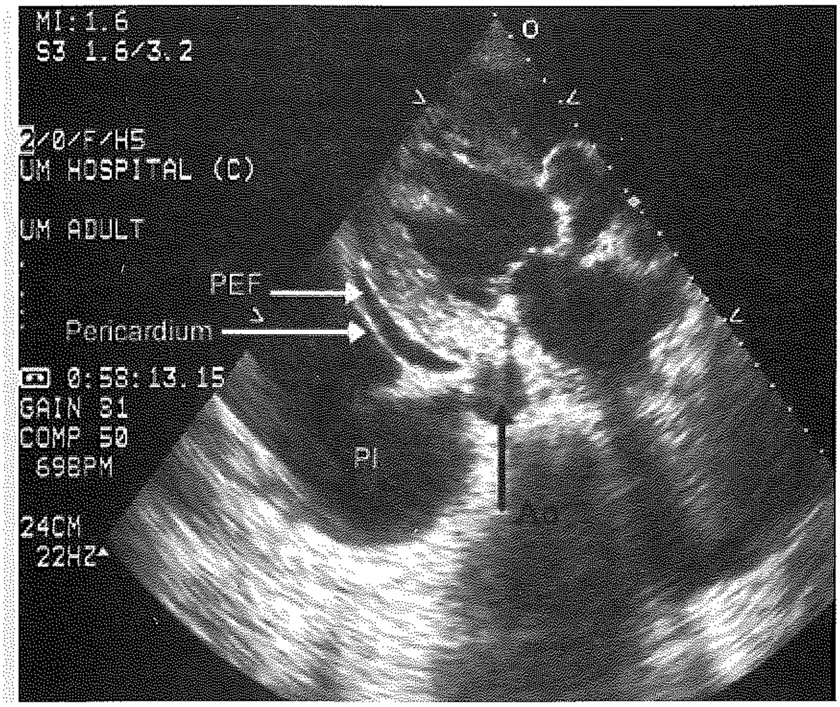
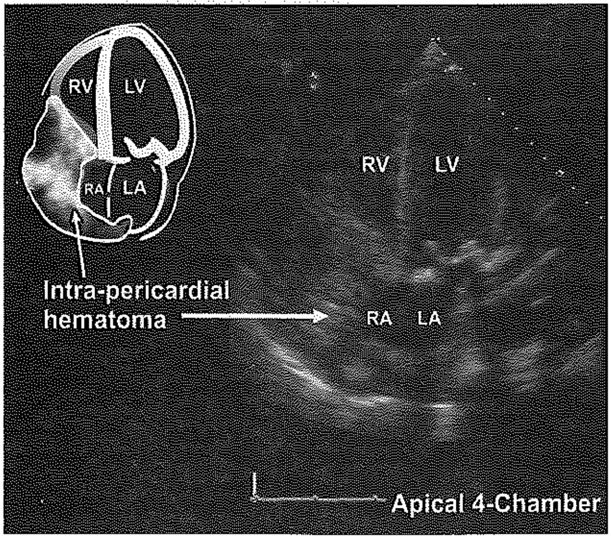


Diagram 169 : PLAX view showing combined case of pericardial effusion and pleural effusion with the pericardial layer in-between & separating the two effusions.
Notice that the pericardial effusion is anterior to the descending aorta in the PLAX.



Post open heart surgery, an intrapericardial hematoma may lead to unexplained clinical deterioration, these hematomas are sometimes missed by TTE, and when suspected TEE is mandatory.

M.mode :

The RV collapse can be detected by the M.mode as shown in the diagram 170.

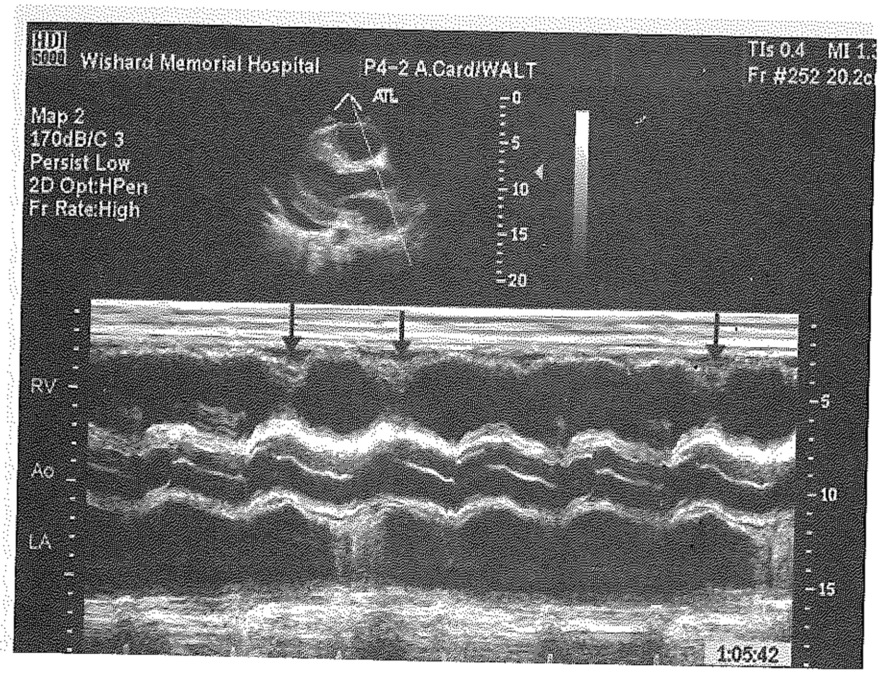


Diagram 170 : showing RV diastolic collapse in M.mode study. The RV collapse is denoted by the arrow

Doppler :

Normally during inspiration the venous return to the left atrium decreases and the venous return to the right atrium increases, so normally during inspiration the trans-mitral flow decreases (15 % reduction) and the trans-tricusped flow increases (25 % increase). In cardiac tamponade there is an exaggeration of this variation. These flow variations can be detected by PW Doppler in an apical 4 chamber view. See diagram 171.

The same concept can be applied for the LVOT VTI and the RVOT flow VTI.

(See before)

N.B : Doppler findings are usually less clinically used in practice because the cardiac tamponade usually present as an urgent condition or as an emergency cardiac situation, and the echocardiograhic study should be done as fast as possible as a bed side echo (emergency echo).

*Cases with increased wall stiffness i.e. LVH, or abnormally high cardiac pressures i.e. pulmonary hypertension, will lead to less evident or absent findings of tamponade by both 2 D and Doppler techniques.

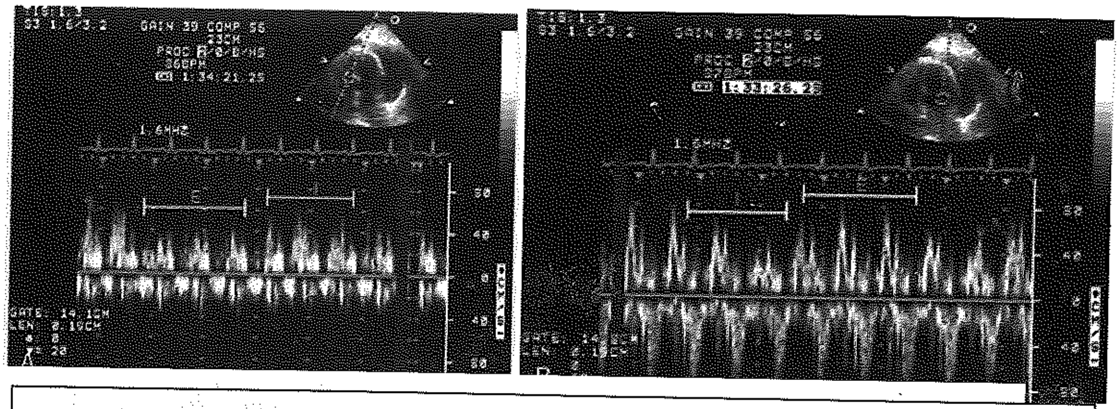


Diagram 171 : Doppler recordings of transtricuspid (A) and transmitral (B) inflow velocities recorded in a patient with a hemodynamically significant pericardial effusion and clinical evidence of cardiac tamponade. The inspiratory (I) and expiratory (E) phases are as noted in the brackets. For the tricuspid valve, note the augmented inflow during inspiration with diminished inflow during expiration. Note the opposite effect seen on the transmitral inflow velocity.

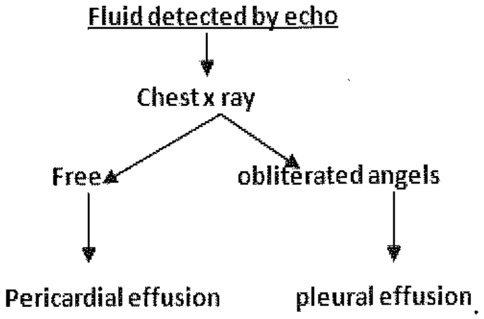
Pitfalls in pericardial effusion and cardiac tamponade:

In difficult echo views, confusion may occur between pericardial effusion and pleural effusion. All the previous methods of differentiation must be thought of before judging, However pericardial effusion is only confused with the left sided pleural effusions, so a chest X ray will be helpful in difficult scans.

A combined case of pleural and pericardial effusion is an easy case because the parietal layer of the pericardium will be seen traversing the fluid. (see diagram 169)

EMSE algorithm in difficult echo views

Remember a combined case will not cause any confusion



*Failure to open any chamber will cause confusion for a beginner sonographer. For example, anticlockwise rotation in the apical 4 chamber view (yet not rotating enough to adjust an apical 2 chamber view) will partially close the RV and a beginner may confuse that for a collapsed RV.

Excessive posterior tilting in the apical 4 chamber view will obscure the LA, and will give a false impression for a beginner that the LA is collapsed.

Parameter	**Constrictive**	**Restrictive**
Atrial size	Normal	Dilated
Pericardial appearance	Thick/bright	Normal
Septal motion	Abnormal	Normal
Septal position	Varies with respiration	Normal
Mitral E/A	Increased (≥2)	Increased (≥2)
Deceleration time	Short (≤160 ms)	Short (≤160 ms)
Anular E'	Normal or increased	Reduced (≤1cm/Sec)
Pulmonary hypertension	Rare	Frequent
Left ventricular size/function	Normal	Normal
Mitral/Tricuspid regurgitation	Infrequent	Frequent (TR>MR)
Isovolumic relaxation time	Varies with respiration	Stable with respiration
Respiratory variation of mitral E velocity	Exaggerated (≥25%)	Normal

The above comparison represents an outline of various parameters which can help in differentiating constrictive pericarditis from restrictive cardiomyopathy. It should be emphasized that in the majority of cases there may be discordant data and that the distinction should be based on the overall appearance and not on a single factor. In complex instances such as combined constriction and restriction following radiation or either entity combined with primary valvular heart disease, many exceptions to these guidelines are anticipated.

Chapter 16 : Assessment of the aorta :

2D echo : the main modality for assessment.

The main pathology the echocardiographer must search for is :

1) **aortic root dilatation**
2) **aortic dissection flap (the clinical scenario will direct the sonographer to search for the flap)**
3) **coarctation of the aorta.**

All parts of the aorta can be examined by 2 D echo.

The ascending aorta can be seen in the high PLAX view (see echocardiography views)

2 D measurements can be taken as in diagram 172.

The descending aorta can be seen in the subcostal descending aorta long axis view (see echocardiography views), and 2 D measurements can be taken as shown.

The ascending aorta, aortic arch and a part of the descending aorta can also be seen in the suprasternal long axis view. See diagrams 172 & 173.

Upper Normal values, these measurements are particularly much more accurate when correlated to the body surface area:

Aortic root : 3cm

Sinus of valsalva : 3.5 cm, 2.1 cm/m^2

Sinotubular junction: 3 cm

Ascending aorta : 3cm

Descending aorta : 3cm , 1.6 cm/m^2

N.B: Revise the guidelines for intervention in aortic root dilatation. Such decisions are beyond the scope of this book.

N.B the most sensitive part for aortic dilatation is the sinus of valsavla, the normal values in adult should be correlated to the body surface area. For clinical practice a value less than 2.1cm/m^2 is normal.

A lot of diseases can either cause or are associated with aortic dilatation at any site of the aorta, also aortic aneurysms can present as the primary disease and can be multiple.

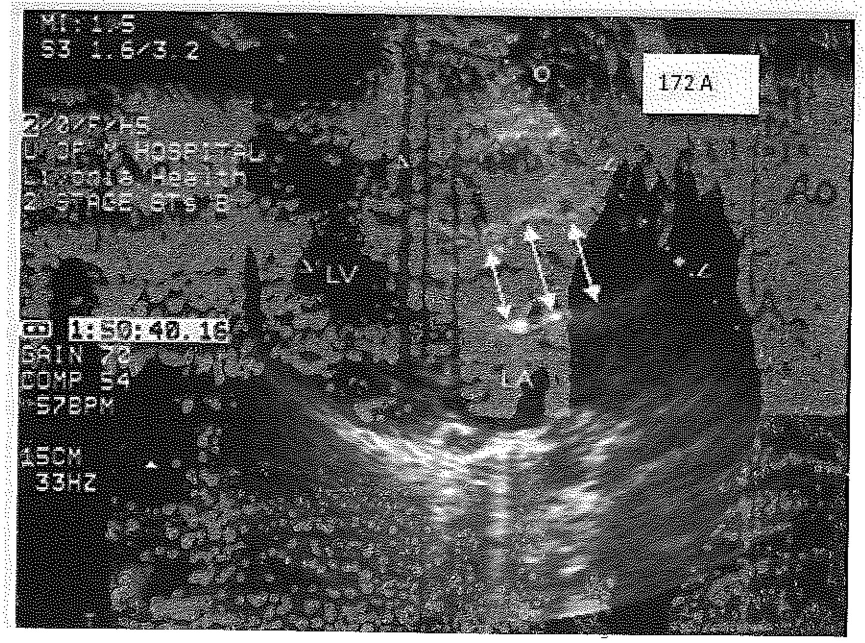
The differentiation between various causes will be made mainly on clinical basis.

Of note is the pattern of the aortic dilatation associated with Marfan syndrome, where the aorta loses its normal contour. See diagram 172 for aortic dilatation, normal and abnormal contours.

Common causes of aortic dilatation:

Age-related aortic dilation
Annuloaortic ectasia
Cystic medial necrosis of the aorta (isolated biscuspid aortic valve or Marfan's syndrome)
Systemic hypertension
Aortitis (syphilis, giant cell arteritis)
Reiter's syndrome
Ankylosing spondylitis
Behçet's syndrome
Psoriatic arthritis
Osteogenesis imperfecta
Relapsing polychondritis
Ehlers-Danlos syndrome

N.B: Bicuspid aortic valve, coarctation of the aorta and aortic dilatation (with increased propensity for dissection) can be associated together. When one abnormality is diagnosed, the sonographer must exclude the other anomalies.



172 A

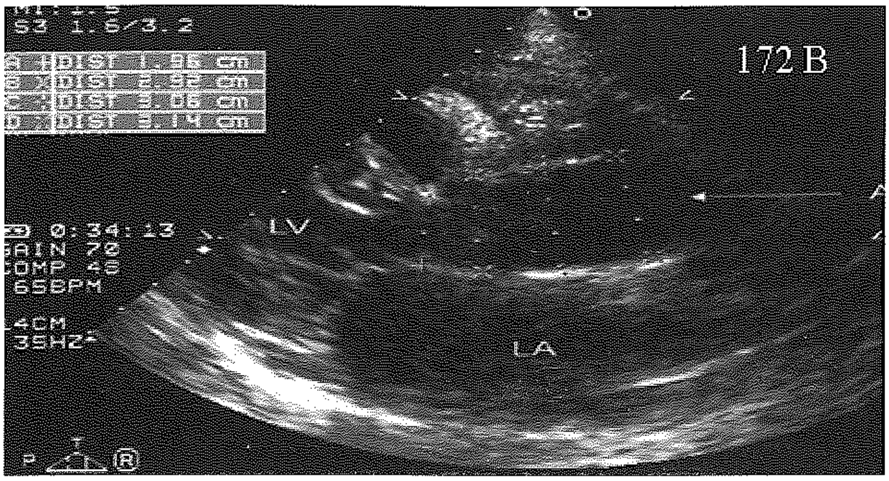


Diagram 172 A &B : exact sites at which the aorta should be measured.

172 A dilated aorta with preserved contour of the aorta, 172 B : dilated aorta with loss of the normal aortic contour.

From proximal to distal : aortic root, sinus of valsalva, sinotubular junction & ascending aorta.

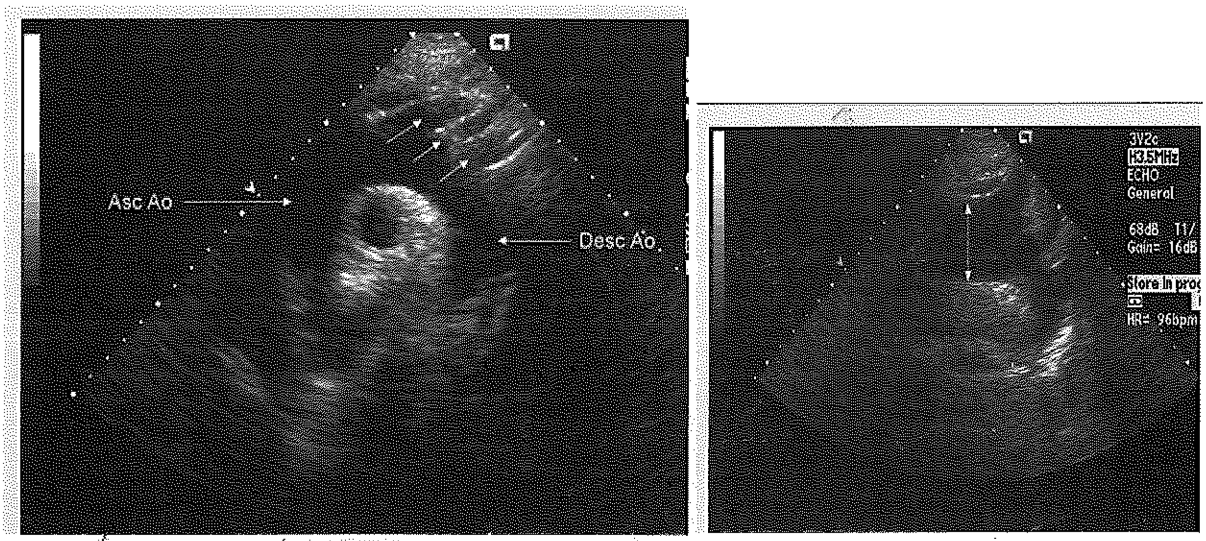


Diagram 173 : measurement of the aortic arch.

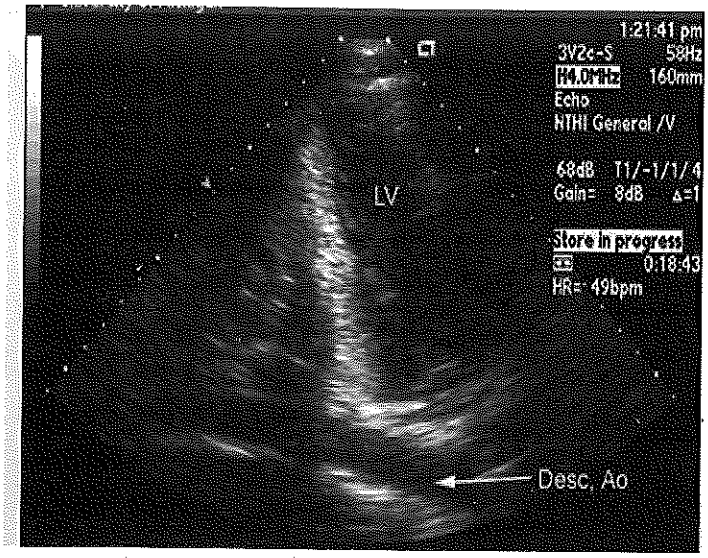


Diagram 174: modified apical view, sometimes with both rotation and tilting motions of the probe, a tube is seen as illustrated above, this is the descending aorta.

Aortic dissection:

The clinical scenario must direct the sonographer's attention to search for the dissection flap. The patient will complain of a sudden onset of severe chest pain. Other risk factors for aortic dissection should not be overlooked, e.g. the age group, presence of aortic dilation or a pregnant female in the 3rd trimester.

2D echo :

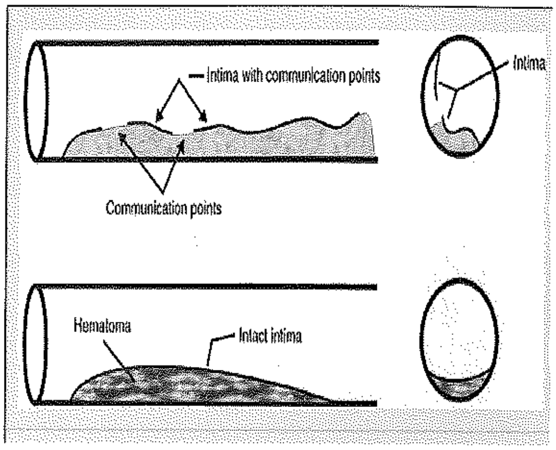
Once suspected, the search for the flap will typically start by the high PLAX view. See diagram 175.

It can be also demonstrated in the PSAX. See diagram 176 .

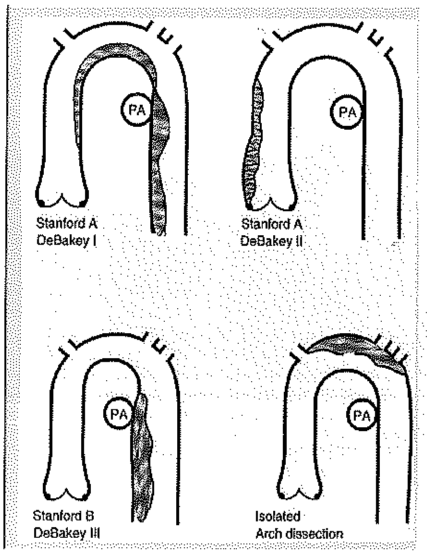
A supra sternal view theoretically should be valuable, but in clinical practice it may be difficult to acquire a clear view in adult patients. See diagram 177.

The dissection flap can be tracked down to the descending aorta in a subcostal view but with lower sensitivity than for the ascending aortic dissections. See diagram 178.

N.B.: Failure to image the dissection flap does not exclude the diagnosis of aortic dissection, and in a high risk patient for aortic dissection with strong positive symptoms & signs suggestive of dissection a TEE can be ordered or even a multi slice CT with contrast.



Schematic representation depicts the forms of acute aortic pathology. A: Depicts classic aortic dissection in which there is a tear of the intima from the media. The column of blood propagates proximally and distally, and there may be multiple communication points between the lumen and the intima media space. B: The spontaneous intramural hematoma variant of aortic dissection in which there is rupture of the vasa vasorum resulting in hematoma in the medial space without communication between the lumen and the hematoma is depicted. The two right-hand schematics depict the same phenomenon in a short-axis view of the aorta.



Schematic representation of categorization schemes for aortic dissection. The schematics include the typical distinction of proximal ascending dissection as well as distal dissection. Additionally, the more recently appreciated isolated arch dissection is likewise depicted. PA, pulmonary artery.

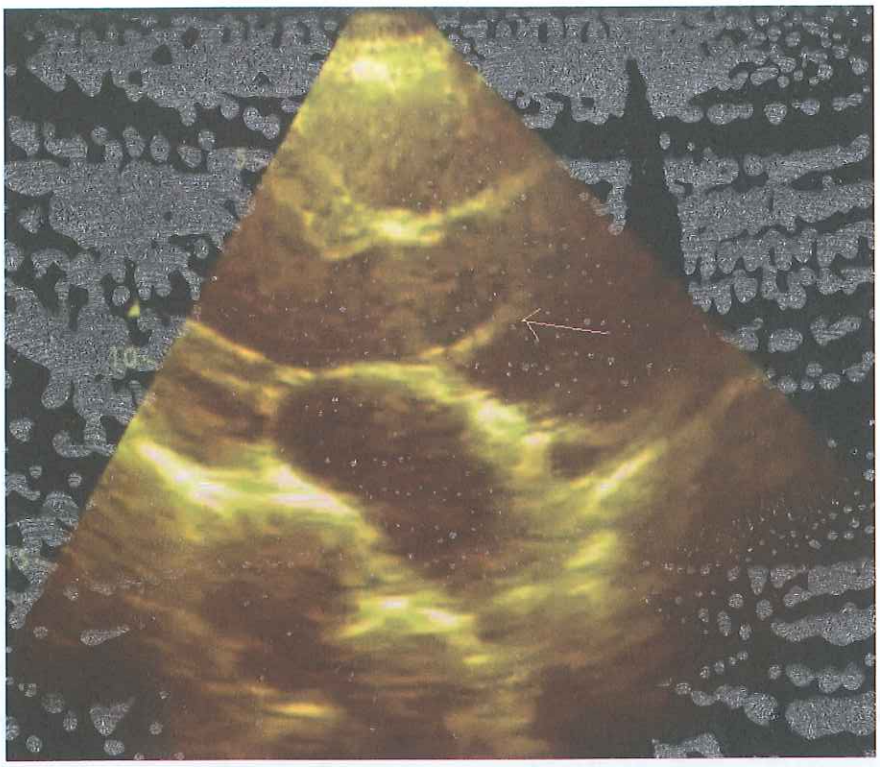


Diagram 175 A : Note the remnants of the intimal flap within the lumen of the dilated ascending aorta (arrows). In diastole, the intimal flap prolapses through the aortic valve into the left ventricular outflow tract. This is one of the several mechanisms for developing aortic insufficiency in acute aortic dissection. Ao, aorta; LV, left ventricle; RVOT, right ventricular outflow tract.

175 B Zoom in PLAX view over the aortic valve and proximal part of the aorta.

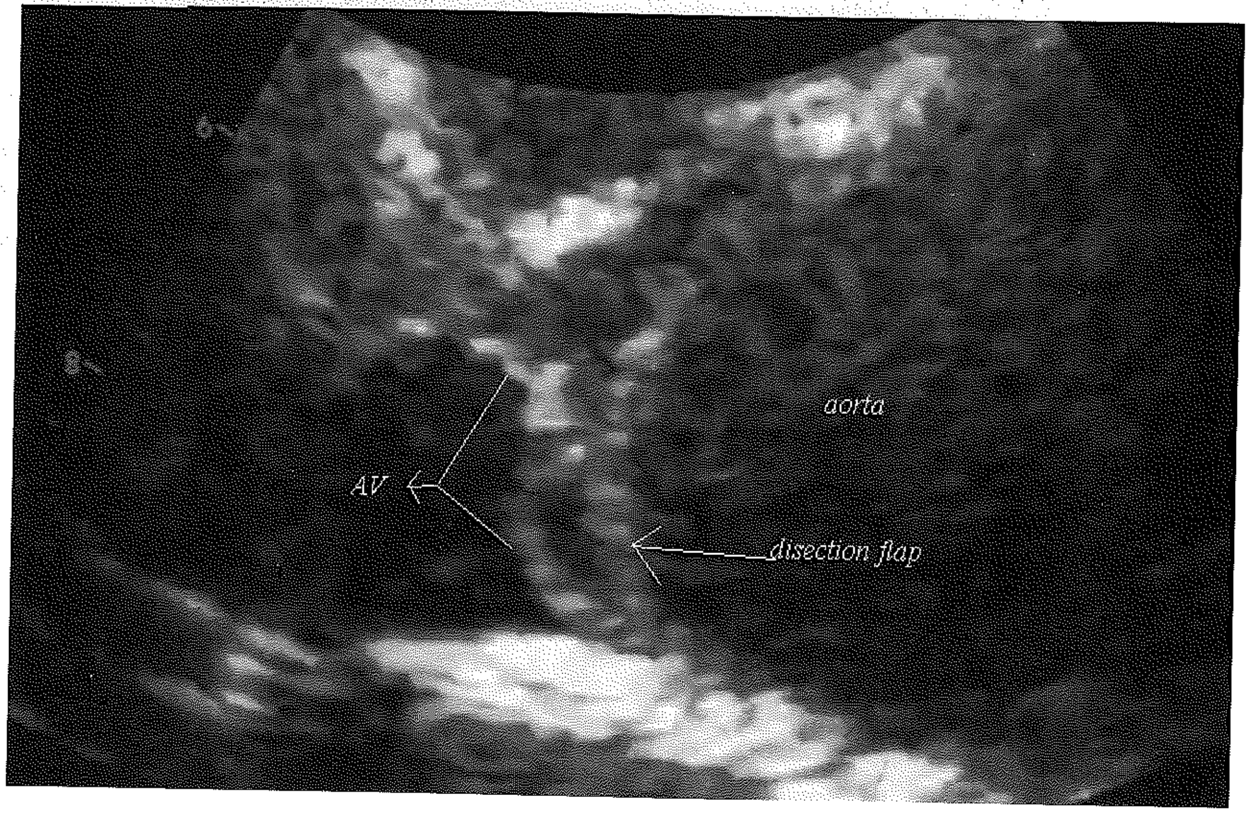


Diagram 176 : showing dissection flap in PSAX.

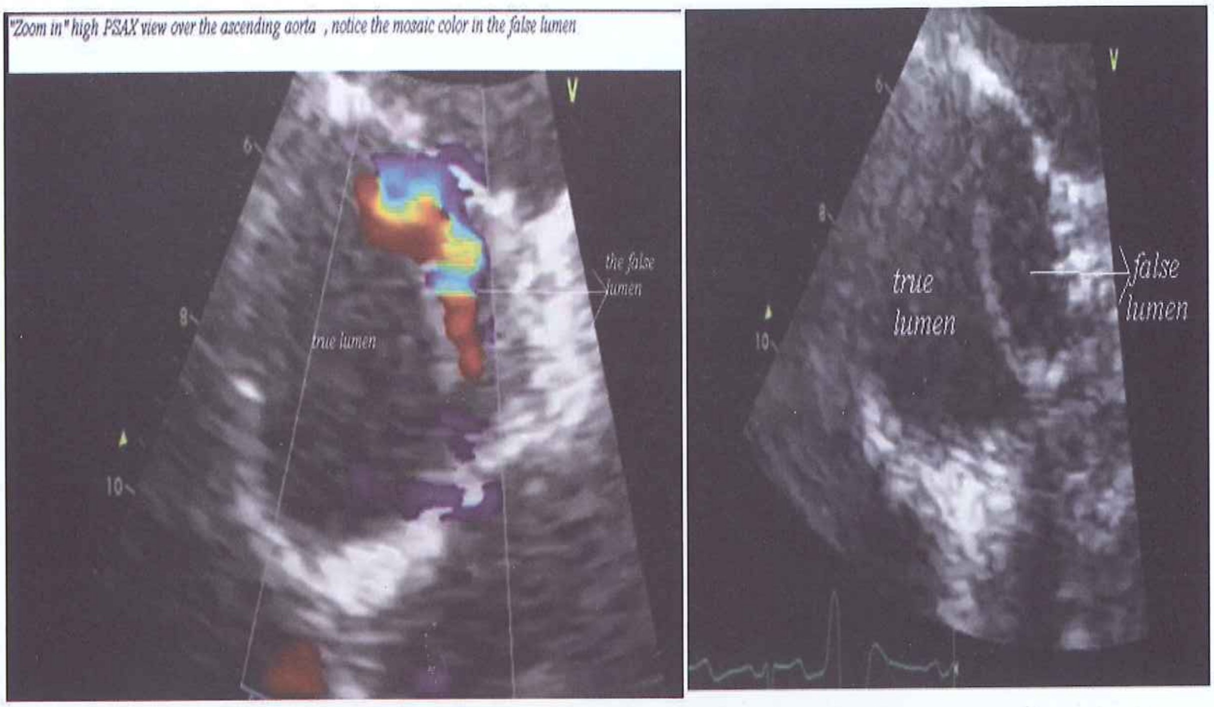

"Zoom in" high PSAX view over the ascending aorta , notice the mosaic color in the false lumen
V
6
8
10
the false lumen
true lumen
V
6
8
10
true lumen
false lumen

Diagram 176 : showing dissection flap in a high PSAX view. Notice the color mosaic inside the false lumen of the ascending aorta.

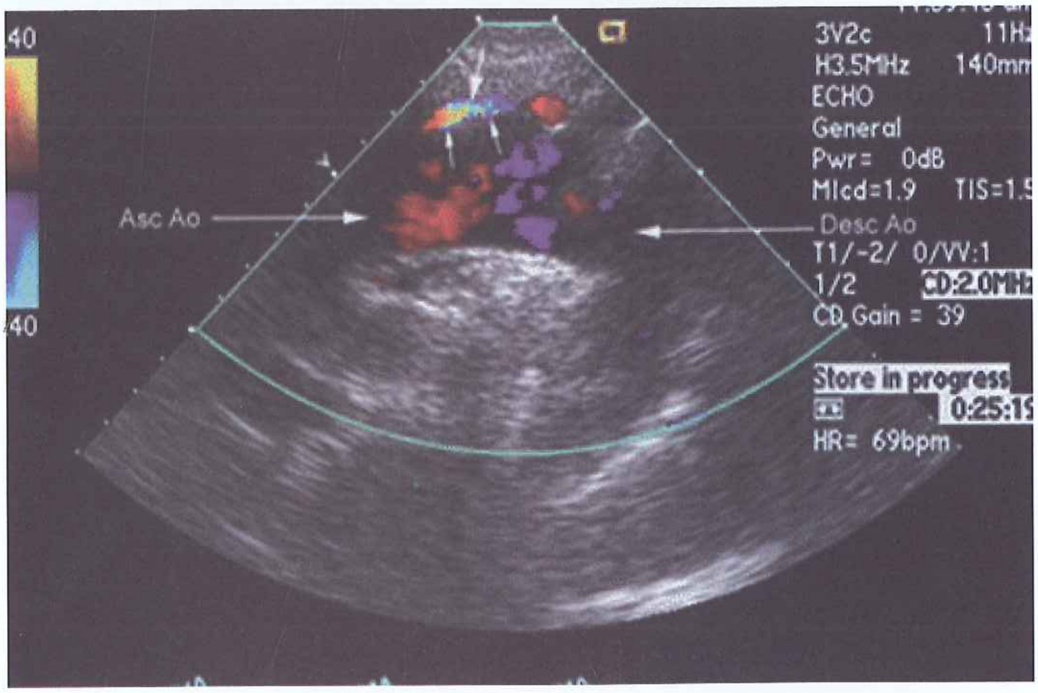
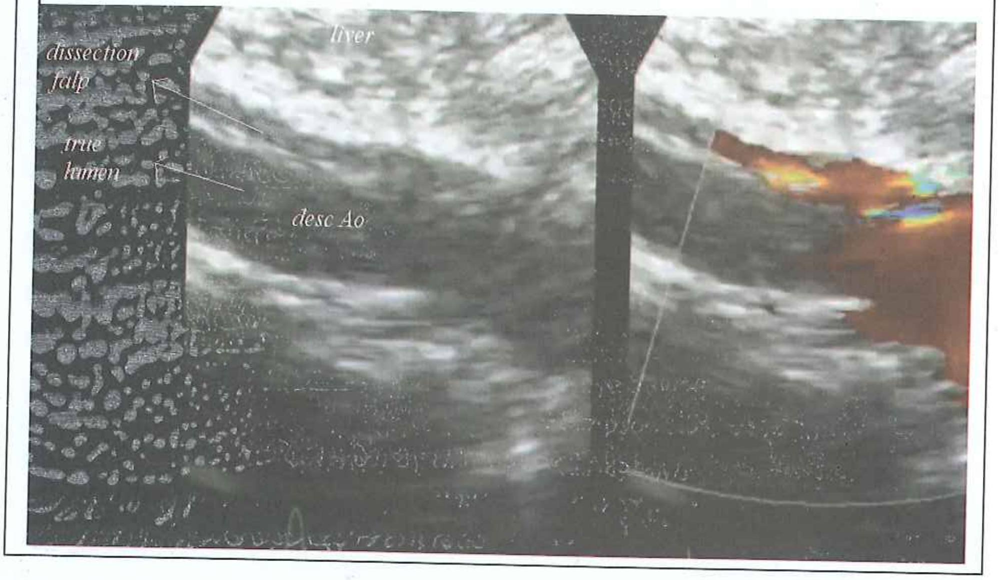


Diagram 177: showing mosaic pattern in-between the layers of the aortic arch, denoting aortic dissection.

Diagram 178 : showing dissection flap in the descending aorta from a subcostal view.

subcostal descending aorta long axis view, (left ward tilting from the IVC subcostal view)



M.mode :

M.mode remains the main technique to identify various layers owing to its better axial resolution.

M.mode can be aligned over the dissection flap for more confirmation, especially if a reverbation artifact is suspected. The reverbation artifact will move with the wall while the dissection flap will move differently from the aortic walls.

Doppler :

Color Doppler can be adjusted over the flap and the mosaic pattern can be seen within the false lumen. See diagrams 176 & 177.

Also it can be used to look for associated lesions / complications, e.g. acute aortic regurgitation.

N.B: Other complications of aortic dissection must be looked for and reported as pericardial effusion.

Cortication of the aorta:

This is a congenital anomaly where there is a stenosis in the descending aorta almost always juxtaductal, i.e. at the site of the ductus arteriosus. See diagram 179.

2D echo : the stenosis can be seen at the anatomical site.

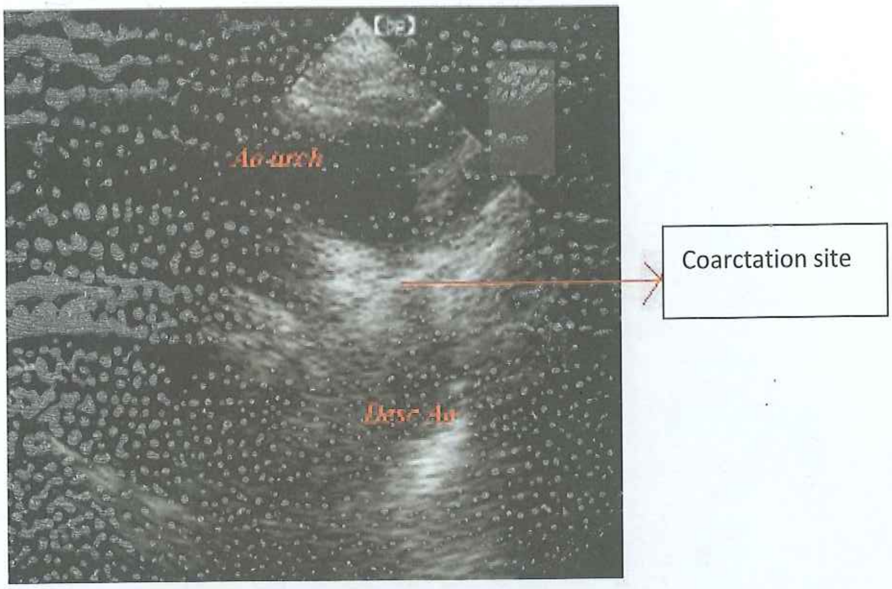


Diagram 179 : aortic cortication , SSLA view.

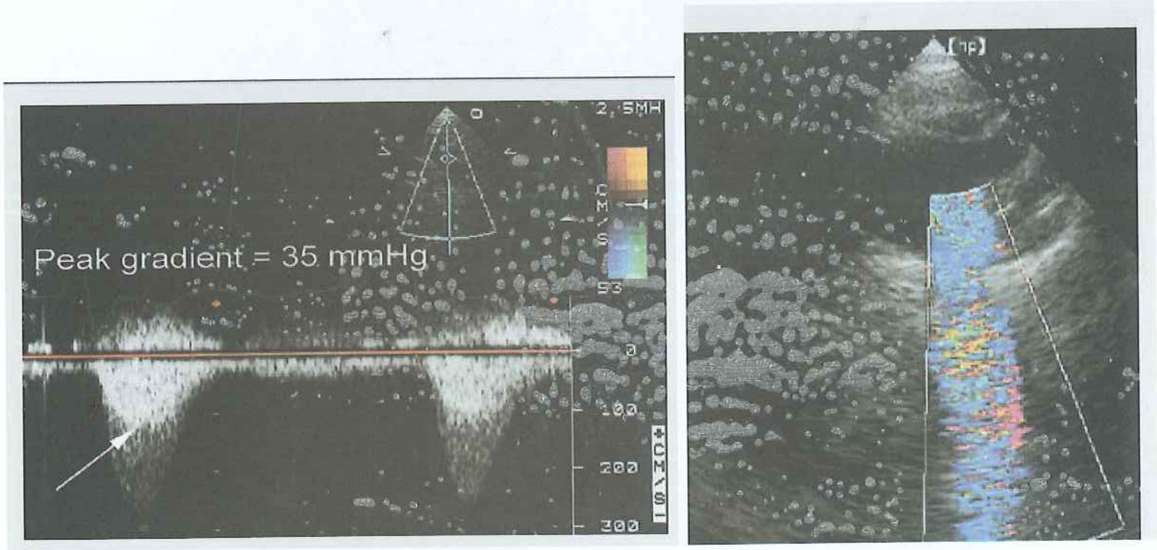


Diagram 180 : Continuous wave Doppler imaging demonstrates a peak systolic pressure gradient of 35 mm Hg across the coarctation. Superimposed within the systolic flow signal is a darker jet (arrow) that corresponds to flow proximal to the stenosis. Note the absence of flow during diastole.

M.mode : no relevance.

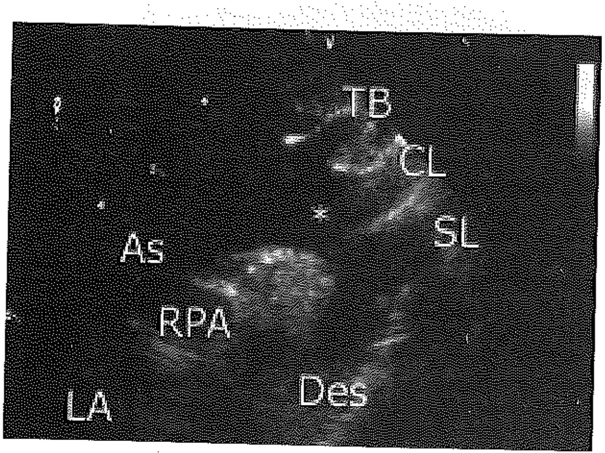
Doppler :

The condition can be diagnosed by aligning CW Doppler on the descending aorta in the suprasternal long axis view to detect the high velocity Doppler signal.

See diagrams 179 & 180: coarctation of the aorta.

See diagram 181 for a normal descending aorta in SSLA view.

Diagram 181: Normal SSLA view



N.B CW derived gradients tend to overestimate the severity of this lesion (due to pressure recovery phenomena). Transcatheter gradients are more accurate.

Color Doppler will show a high velocity turbulent mosaic pattern at the anatomical site.

Chapter 17: Prosthetic valves:

In this chapter we will only discuss the various echocardiographic findings that can be seen with well- or malfunctioning prosthetic valves of various types, in addition to any relevant information that aids understanding.

Before assessment:

The echocardiographer must know the full data about the prosthesis and the type of operation performed.

Data that has to fulfill before examination is: the type of the valve, the size of the valve, the baseline gradients before hospital discharge (preferred a copy of the surgical operation document including full details), the year of manufacturing, the number of valves replaced, and the reason for replacement.

Any missed data may lead to an inaccurate clinical decision making, for example one finding can be accepted to be observed over one prosthesis while considered abnormal for another prosthesis as we will discuss later.

GENERAL CLASSIC ASSESSMENT:

Quantified assessment of prosthetic valves is the same as for a native valve with special considerations. 2D, M.mode, and Doppler will be used for full analysis. 2 D imaging, i.e. the shape of the prosthesis, will vary widely according to the valve type, M.mode will be of little value and only used when it seems rational (in special situations), and PW & CW Doppler will show nearly the same waves as a native valve with some variations. PHT derived mitral valve area are still validated with tendency for slight overestimation of the actual valve area. Color Doppler will be the main modality to quantify for a regurgitation jet &/or para-valvular leak.

2D echo :

Shadowing and reverberation artifacts are also classically seen behind all types of prosthesis (excluding homo & autografts). The shape of these reverberations will vary according to the valve type, as we will discuss later in the 2 D examination of the various types of prosthetic valves.

Surgical sutures are commonly seen attached over the prosthetic material, and sometimes it is so difficult to differentiate them from vegetations either by TTE or even by TEE examination, especially if the clinical situation mimics a case of infective endocarditis. Huge echocardiographic experience is needed and/or a second opinion is taken to differentiate a suture from vegetation.

Doppler:

A mild to moderate increase in the pressure gradients can be classically observed over all prosthetic valves (excluding homograft and auto graft), and the accepted amount in increase is determined by the manufacturer according mainly to the valve type and size.

The presence or absence of E/A ratio will depend on the physiological function of the LA rather on the valve type, i.e. the A wave is preserved in sinus rhythm but lost in AF.

Classically, Color Doppler will show mosaic pattern flowing out of the prosthetic valve orifice (excluding homo & auto grafts). Also mild transvalvular leaks are classically seen (These jets are important to backwash the valve aiming to decrease thrombosis incidence)

In minor cases with a clinically free patient small jets of paravalvular leaks can be considered as insignificant.

Types of valve substitutes:

Autograft "Ross procedure" :

Replacing the aortic valve including part of the ascending aorta with the patient's own pulmonary valve. This type of operation is almost always limited to pediatric surgery. (Reimplantation of the coronaries is mandatory and dealing with the pulmonary valve varies from one surgeon to another)

Homograft:

This is only done for the aortic valve; a cadaveric "cryo preserved" aortic valve including part of the ascending aorta is used for aortic valve replacement (and the antigenic material is removed).

Echo findings are for these two valves are similar.

2D echo : the valve will appear as if native. The only clue may be the sternotomy scar !

Some systolic fluttering may be noticed, and some minor sclerocalcifications may be seen.

M.mode : as in native valve.

Doppler : as in native valve.

Assessment of stenosis and regurgitation lesions will be done exactly as in native valve.

Bioprosthesis :

A bioprosthetic valve includes both prosthetic and biological materials. A bovine pericardium or porcine leaflets are used as the leaflet material and stretched over a swing ring. See diagram 182A.

These valves can be used for mitral, aortic and tricuspid positions.

2 D echo: see diagram 182. Notice the unique shape of the valve, notice the reverberations and shadowing artifacts.

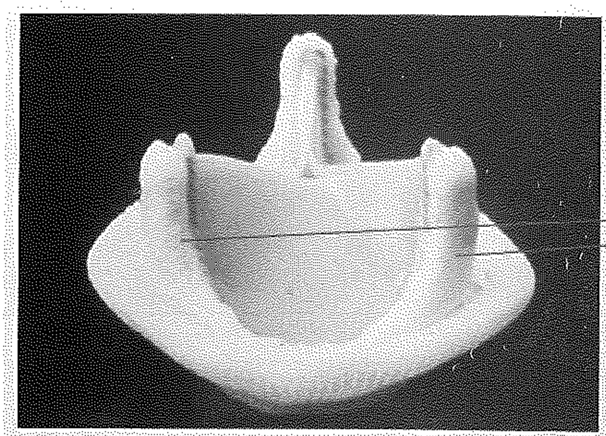


Diagram 182 A : a prosthetic porcine valve.

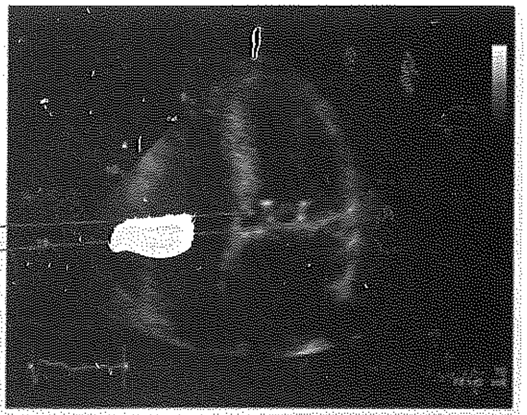


Diagram 182 B : echocardiographic imaging of porcine bio prosthetic valve.

M.mode : of little value.

Doppler: this is the same as in native valves but with relatively increased pressure gradient. See diagram 183. Color Doppler will show aliasing.

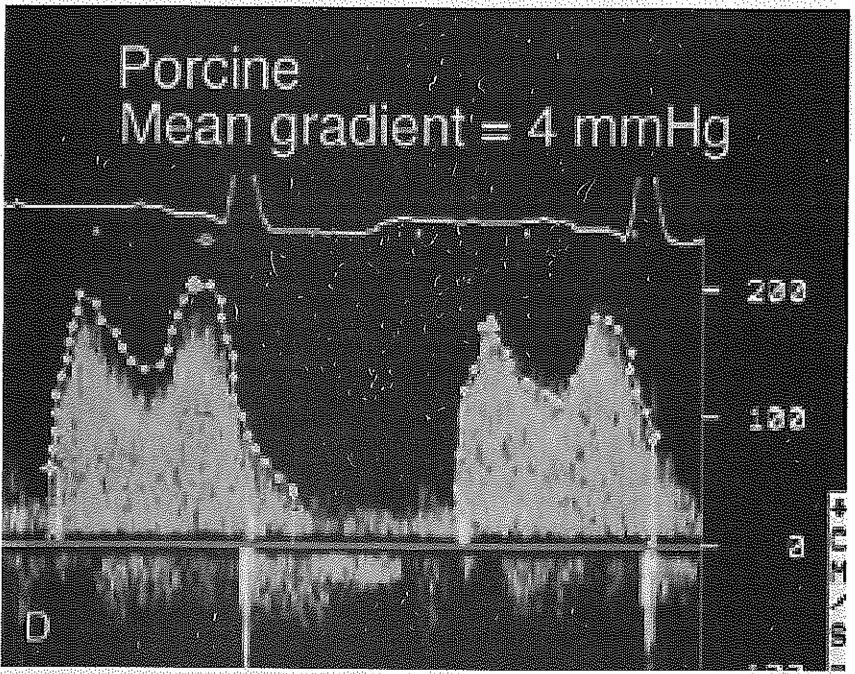


Diagram 183 : Doppler study of a porcine valve in the mitral position.

Commonly used mechanical Prosthetic valves:

Bi-leaflet tilting discs : used in both mitral and aortic positions and uncommonly in the tricuspid position.

This valve is formed of a ring with two tilting discs as shown in diagram 184.

These leaflets move passively by pressure gradient.

2 D echo : see diagram 184. Notice the specific shape of the valve, notice the reverberations and shadowing artifacts.

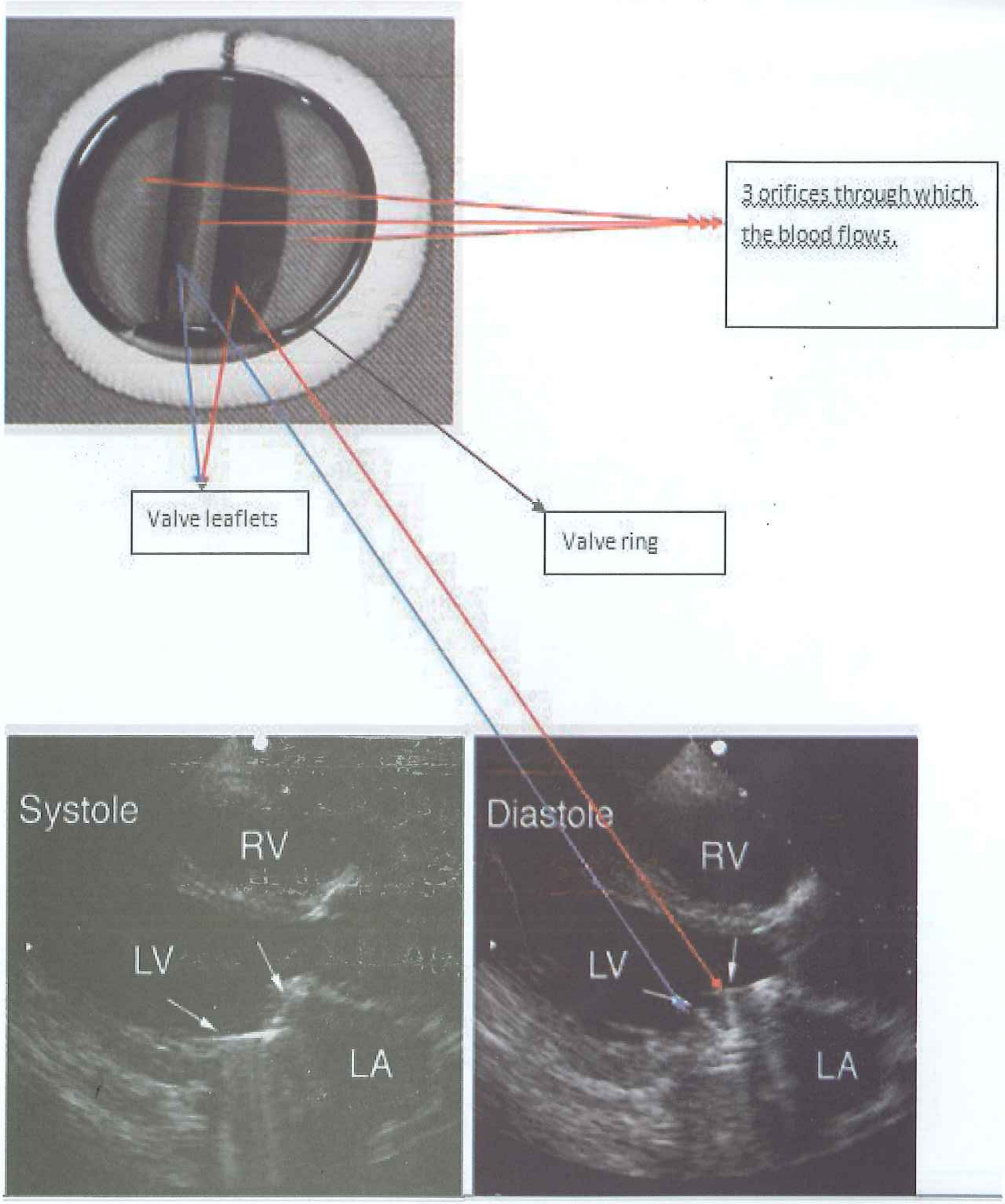


Diagram 184 : A bileaflet tilting valve

M.mode : less commonly used.

Doppler: see diagram 185. Notice the opening and closing clicks shown in the Doppler signal.

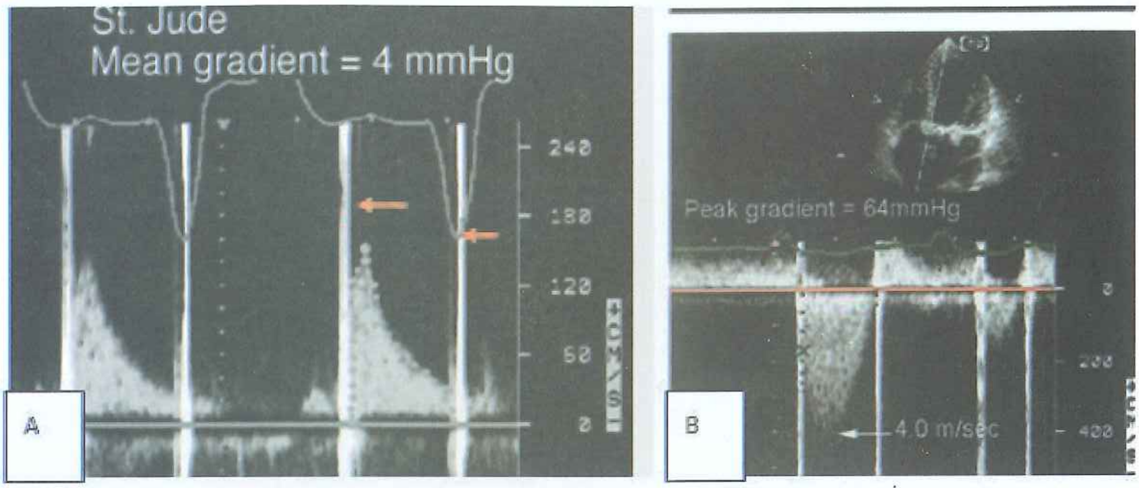


Diagram 185 A & B : Doppler study on a bi-leaflet tilting disk valve. Notice the closing and opening clicks (denoted by arrow) on the Doppler study. A : mitral position, B: aortic position. Notice the classic increased velocities over the prosthetic valves

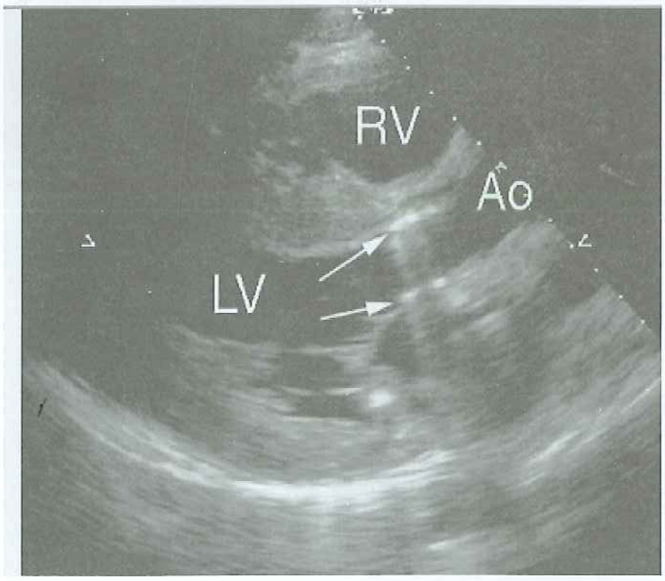


Diagram 186 : Prosthetic Bi-leaflet tilting valve in the aortic position.

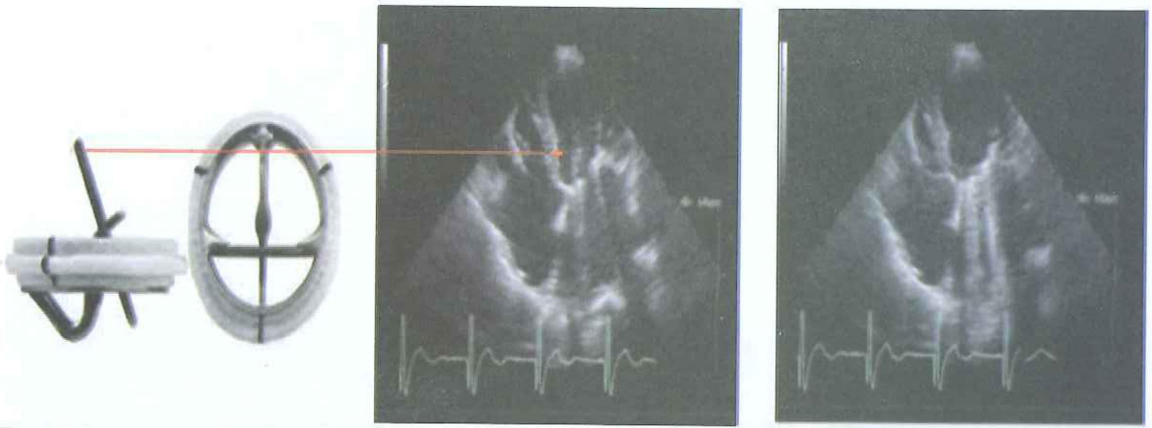
Single leaflet titling disc:

This valve is formed of a ring with one tilting disc as shown in diagram 187.

It functions as the bileaflet titlting valve. (bileaflet tilting valves have a better heamodynamic profile)

2 D echo : see diagram. notice the different shapes of the valve, notice the reverberations and shadowing artifacts.

Diagram 187 : single leaflet tilting valve.



Diastole (the valve is opened)

systole (the valve is closed)

M.mode : less commonly used.

Doppler: C.W doppler as before.

Color Doppler can be used to search for classic transvalvular regurgitation jets, or pathological para valvular leaks. See diagrams 188 & 189.

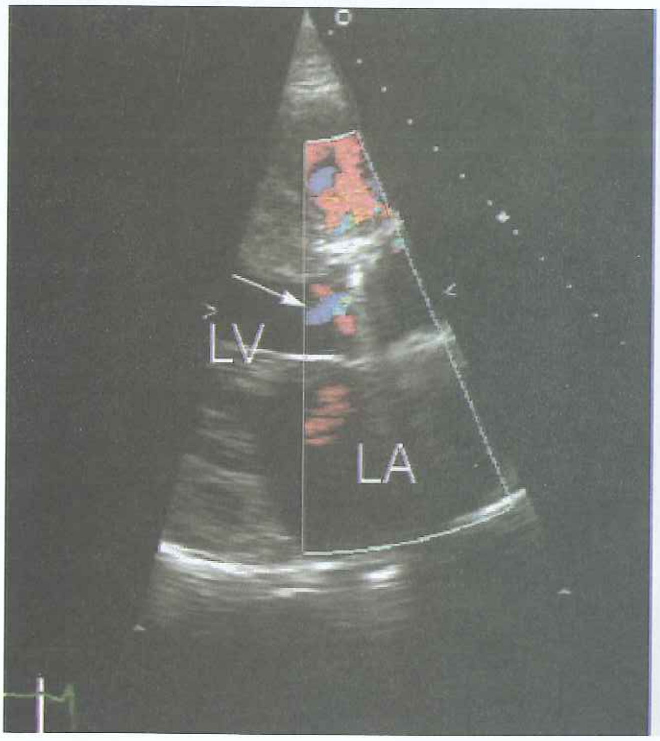


Diagram 188: color Doppler on a prothetic aortic valve in a PLAX view; Physiologic regurgitation through a St. Jude aortic valve is shown in the aortic position. The jets originate at the periphery and appear to

cross just below the valve (arrow). The occurrence of this type of regurgitation is part of the design of many prosthetic valves. LA, left atrium; LV, left ventricle.

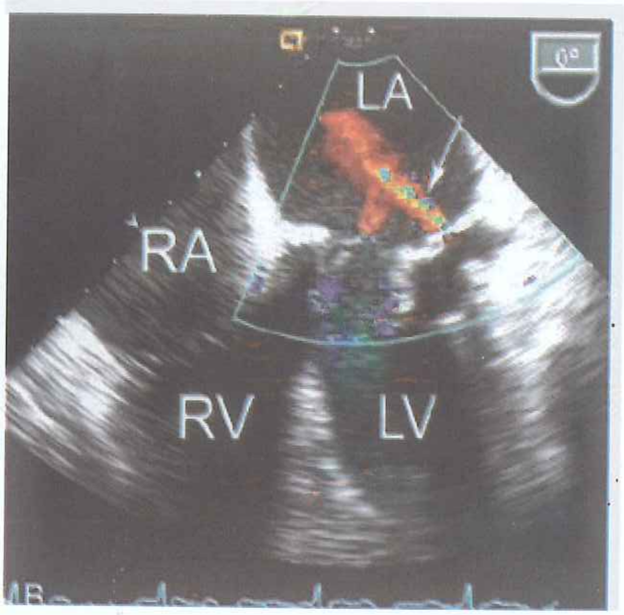


Diagram 189: TEE study (apical 4 ch view equivalent) Color Doppler imaging demonstrates both trans valvular and paravalvular (arrow) mitral regurgitation.

N.B. Based on our experience in echocardiography training, a beginner echocardiographer may confuse a single leaflet tilting valve for a bi-leaflet tilting valve with one malfunctioning leaflet or vice versa.

This needs experience! Nevertheless, increased pressure gradients over a prosthetic valve over its set pressure gradient aids differentiation whether it is a single leaflet prosthesis or a malfunctioning bileaflet prosthesis. Accepted gradients over various types of prosthetic valves according to the manufacturers should be revised from each patient's card.

The main challenge the echocardiographer faces is to judge whether the prosthetic valve is well functioning or malfunctioning. The challenge is more difficult when the clinical situation is critical for example patients in fever, acute dyspnea , pulmonary edema or shocked patient.

TEE is indicated in most cases of prosthetic valves with any suspected pathology, to confirm the diagnosis and to search for other complications.

The echocardiographer should dedicate the study to search mainly for the following abnormalities:

1) Masses
2) Abnormal valve motions: a) rocking motion. B) stucked leaflets.
3) Abnormally increased pressure gradients (more than those mentioned by the manufacturer)
4) Regurgitation jets/ paravalvular leaks.

1) MASSES:

TEE has much higher sensitivity and specificity to detect a mass in the setting of a prosthetic valve.

2 D echo : the mass should be described in terms of its size, shape, surface, attachment, echo density, motion, location.

Take into account that a vegetation is an infected thrombus, so differentiation between both should be done on clinical basis, i.e. with low INR values (mechanical prosthetic valve) the mass is most probably a thrombus, while in a feverish or toxic patient it is mostly a vegetation.

No single modality can differentiate a pannus from a thrombus (pannus is a chronic condition in which the fibrous tissue creeps over the base of the valve hindering its normal opening &/ or closing). So differentiation between both can be made on clinical basis. With acute symptoms the mass is likely to be thrombus/ vegetation, while with chronic symptoms the mass will be mostly a pannus.

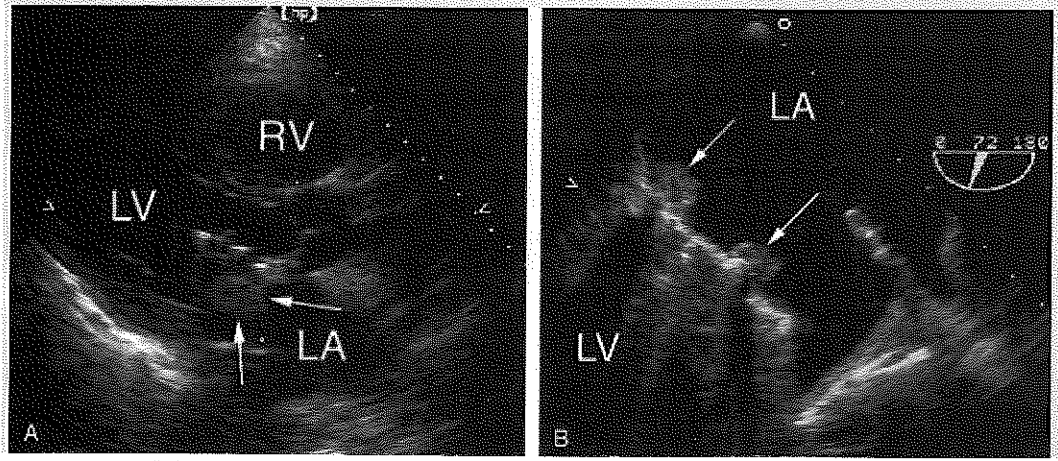


Diagram 190 : A : PLAX showing a mass over the mitral valve, B : TEE study (apical 2 ch equivalent view) in the same patient showing 2 masses denoted by the arrows.

M.mode : though not routinely used, M.mode can yield further information about the nature of the mobility and the relationship of the mass with the surrounding structures especially in apical masses.

Color Doppler : the use of color Doppler can yield surprising findings about the nature of the mass, for example color flow Doppler showing a flow inside a mass suggests a vascularised tumor, e.g. Myxoma. However a myxoma mostly has a pedical attached to the IAS. Also color Doppler will diagnose any para valvular leaks aiding the diagnosis of infective endocarditis.

2) ABNORMAL MOTION OF A PROSTHETIC VALVE:

A. EXCESSIVE RING MOTION "ROCKING MOTION":

This is suggestive of a dehiscent valve due to a slipped suture. This could happen as a result of poor surgical technique or as complication of infective endocarditis.

2D echo : the valve ring can be visualized with abnormal up and down rocking motion (needs experience).

M.mode: of little value.

Color Doppler : The dehiscent valve could be confirmed by the presence of a paravalvular leak by color Doppler. A TEE will also aid the diagnosis.

B. STUCKED VALVE :

Any abnormal mass can interfere with the normal motion of the leaflets, up to completely hindering the valve from opening, a condition known as a stucked valve.

2D echo : visualize the stucked valve as a fixed leaflet. Also the reverberation artifact can sometimes aid the diagnosis by being fixed artifacts instead of showing simultaneous motion with the valve.

M.mode : less commonly used.

Doppler :

CW Doppler: will show high velocity jet with increased PPG and MPG, PHT can still be used for mitral valve area calculation with slight tendency for over estimation of the actual area. See diagram 171.

N.B : Any prosthetic valve may stuck in the open position yielding less severe P.G across the valve.

N.B : The classic click heard during the clinical auscultation may be present with the stuck valve.

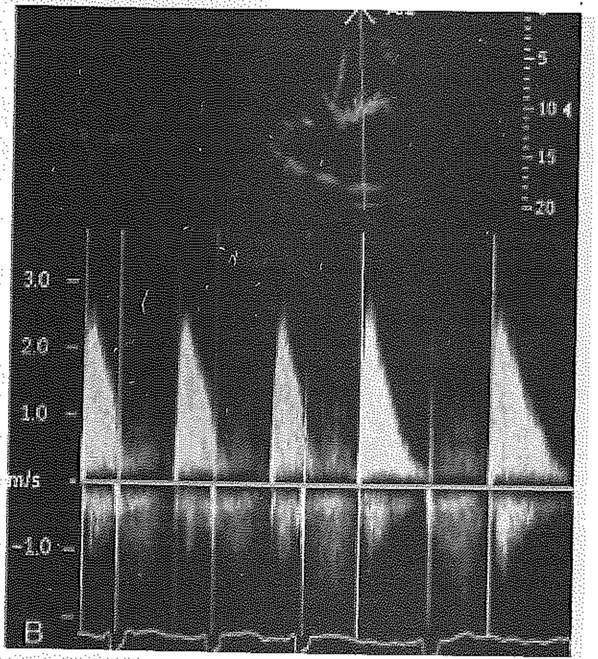


Diagram 191: Shows increased velocity by CW Doppler over a prosthetic mitral valve. The velocity is encroaching 3 m/s. Classic calculations (MPG, PHT) can be used as before.

3) ABNORMALLY INCREASED PRESSURE GRADIENTS:

Doppler study is the only modality to quantify pressure gradients:

CW Doppler should be aligned exactly as in native valve and the Doppler signal can be analyzed (see stenotic lesions)

Color Doppler : an exaggerated high velocity jet will be seen flowing from the prosthetic orifice into the chamber.

Of notice is that the mitral valve may stuck in a way that the flow is directed towards the LVOT. See diagram. Extreme care should be taken in this situation.

4) REGURGITATION JETS/ PARAVALVULAR LEAKS:

Color Doppler is the main modality to diagnose paravalvular leaks.

By adjusting the color Doppler upon the needed valve and its relevant chamber into which the regurgitation jet/ paravalvular leak will occur. The condition can be diagnosed with technical difficulties. These technical difficulties are mainly with the mitral prosthesis where the regurgitation jet / para-valvular leak is over masked by the reverberation artifacts. Moreover ghost artifacts are very common to occur inside the LA with prosthetic mitral material.

Differentiation whether this is a ghost artifact or a true pathological jet will depend on the sonographers experience and the criteria of the true jet described previously in " regurgitation lesions"

See diagrams 192 and 193 to compare ghosting artifacts with true regurgitation lesions.

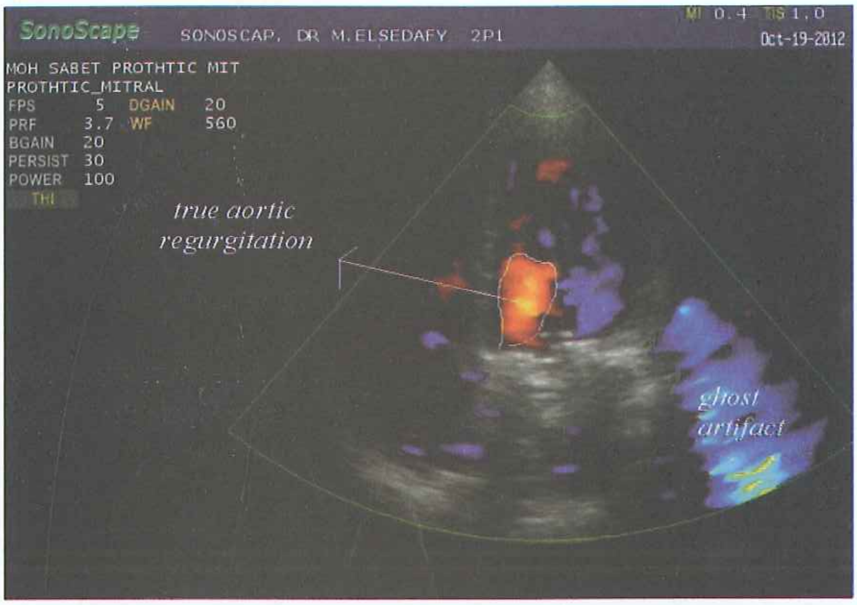


Diagram 192: A ghost artifact. Notice the sharp borders of the color jet. In a playing loop, this color mosaic will appear as flashes, known as color ghosting artifacts. Also, the ghost artifact appears out of the clinical scenario and does not respect the anatomical boundaries. The main dilemma is when this ghost fills the left atrium. It may be mistaken by a beginner echocardiographer as mitral regurgitation, CW Doppler will be very convenient in discriminating both conditions.

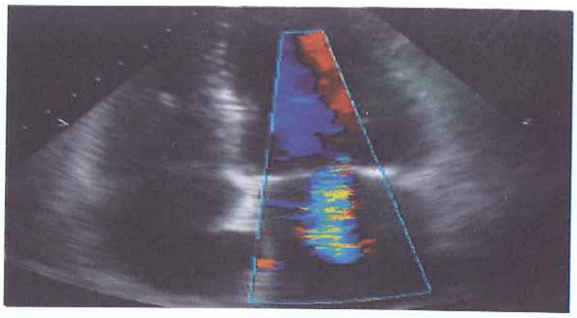


Diagram 193: Regurgitation lesion. Notice the ellipsoid shape of the jet. In a playing loop this color mosaic will have time corresponding to the systolic time.

N.B : When suspicion is raised about the diagnosis of a regurgitation jet by color Doppler, CW can definitely confirm the diagnosis, by aligning the CW over the color Doppler “suspected” jet, a high velocity dense Doppler signal will be displayed in cases of true jets, while in ghost artifacts no such signal will occur.
See diagram 192 for ghost artifacts and true jets.

Chapter 18: Intra cardiac masses and tumors.

The diagnosis of the origin of a mass on the basis of 2 D echocardiography will always remain a source of conflict and misinterpretation. However the echocardiographer experience in addition to the clinical scenario will always be the main 2 key points for diagnosis.

Differentiating a vegetation from a thrombus : remember that the vegetation is an infected thrombus !

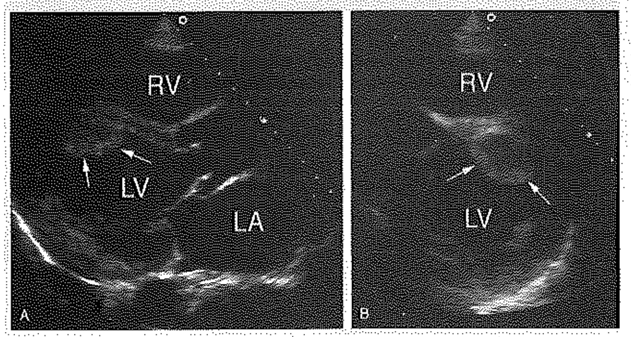
All the factors suggested by different echocardiography laboratories only gives criteria for suggestion, and the final diagnosis is made according to the clinical condition.

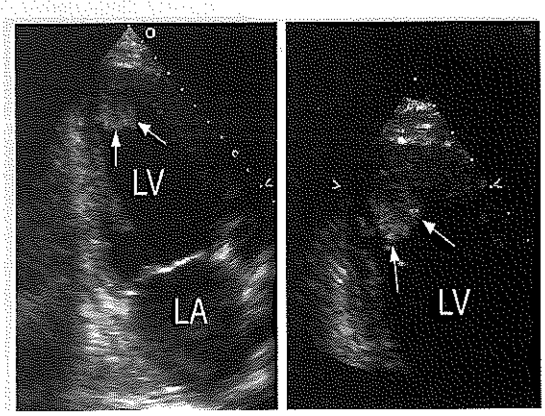
An example : in the setting of mitral stenosis, a LA mass seen by echo is most probably a thrombus, also, in the setting of a prosthetic mitral valve with signs of acute dyspnea and the absence of any toxic symptoms and signs, a mass seen by echocardiography over the mitral valve is mostly a thrombus too, leading to a condition called stuck valve.

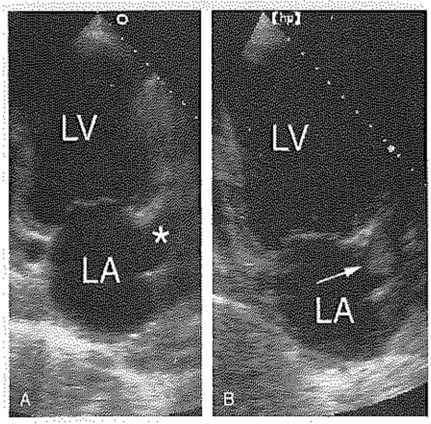
On the contrary, in the presence of any toxic symptoms &/ or signs, a mass seen in the heart will be reported as a vegetation especially if a risk factor for infective endocarditis (IEC) is present.

Factors suggesting a vegetation includes any mass with the following criteria:

- ✓ Associated risk factors or the mass is in the path of a high velocity jet.
- ✓ Irregular in shape
- ✓ Attached to the upstream side of the valve.
- ✓ Relatively Low echo-dense.
- ✓ Mobile and /or oscillating







Examples of a left ventricular mural thrombi (arrows) visualized in vicarious views and in different patients.

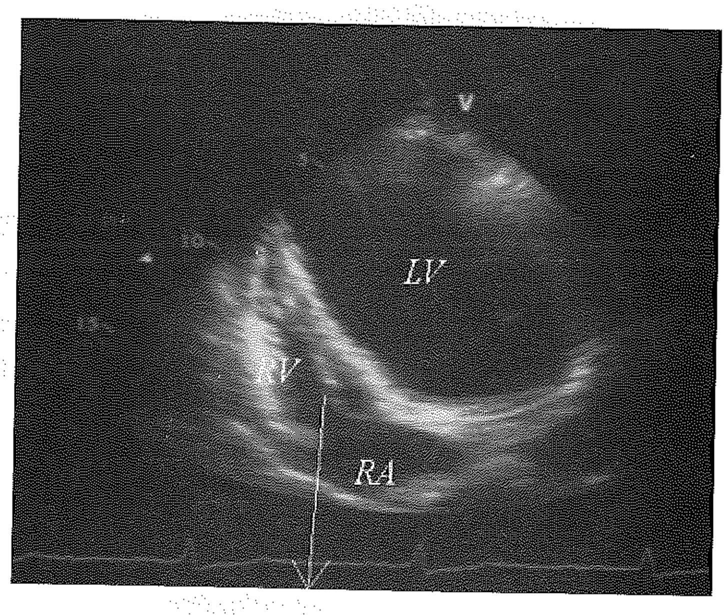
Normal masses :

Normal RA masses include :

- Eustachian valve (see diagram 137 & 138)
- Chiari network (see diagram 139)
- Crista terminalis (see diagram 140)
- Catheters/pacemaker leads (see diagram 195)
- Lipomatous hypertrophy of interatrial septum (see diagram 196)
- Pectinate muscles (mainly seen by TEE exam)
- Fatty material (surrounding the tricuspid anulus)

Normal RV masses:

- Moderator band (diagram 197).
- Muscle bundles/trabeculations .
- Catheters and pacemaker leads (see diagram 195).



A pace maker lead

Diagram 195.an angulated Ap.4 ch view to image the pacemaker lead

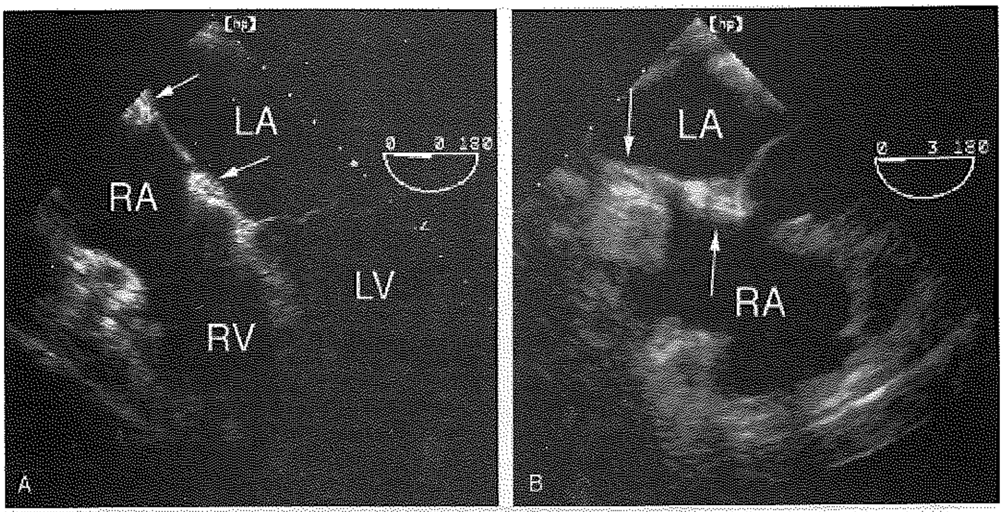


Diagram 196: Lipomatous hypertrophy of the IAS shown by the arrows. A: in an apical 4 chamber view equivalent (TEE study), B: angulated view.

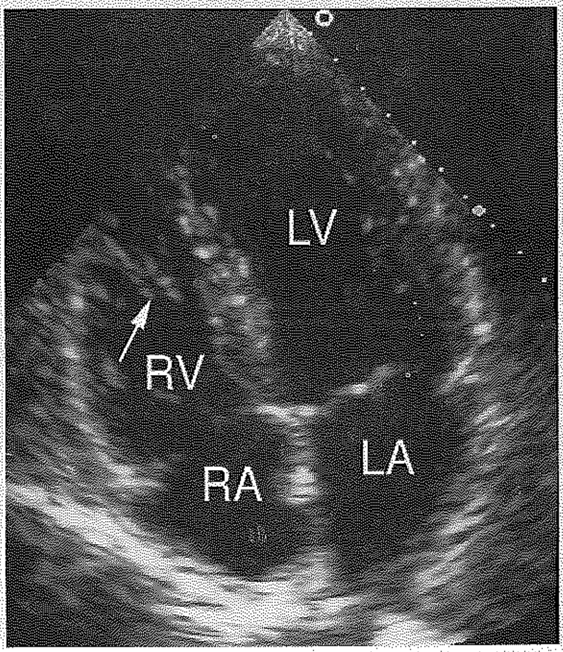


Diagram 197: Showing moderator band, muscle band joining both RV walls.

Normal LV masses :

- False chords (tendon) (see diagram 198).
- Papillary muscles (see diagram 199).
- LV trabeculations (see diagram).

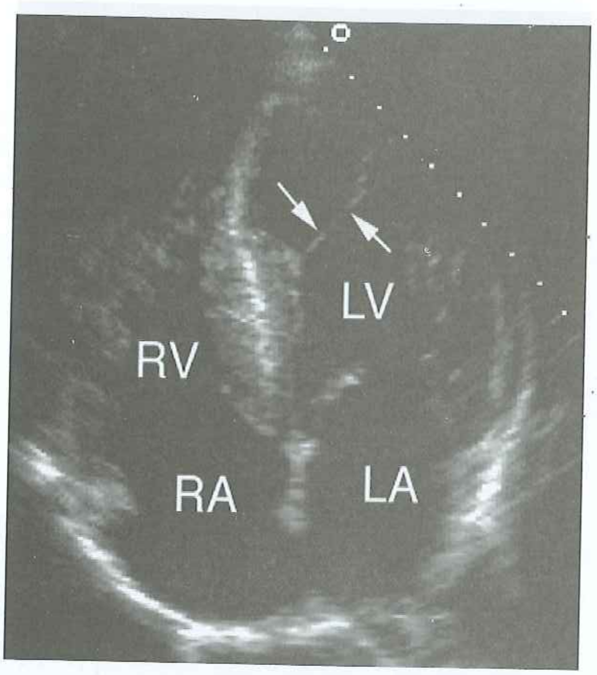


Diagram 198 : false chord shown by the arrows.

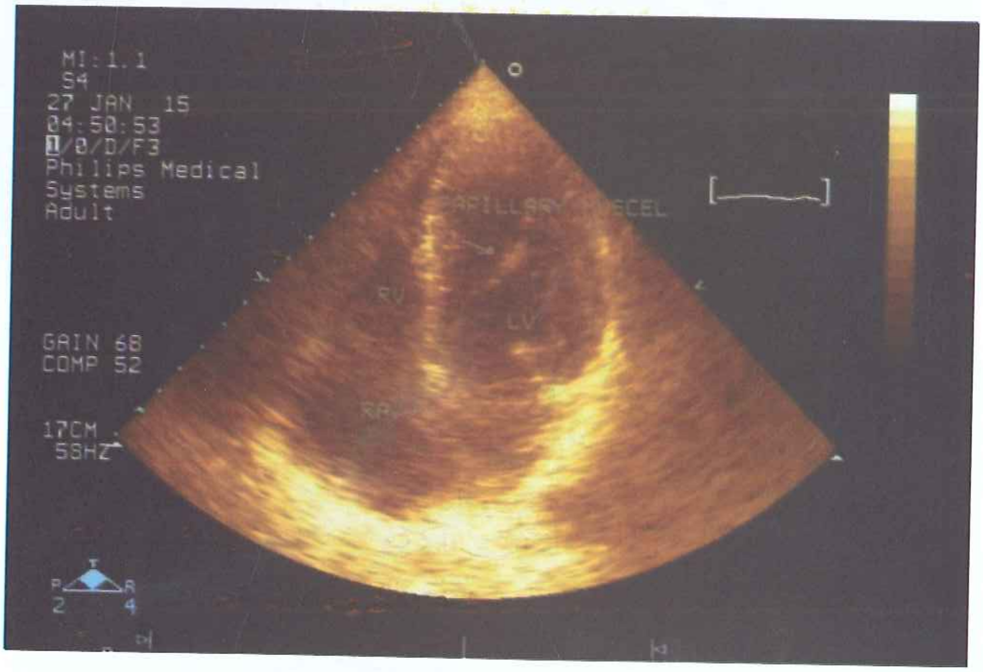


Diagram 199: showing papillary muscle.

Abnormal intracardiac masses :

Most cardiac tumors are secondaries. If primary, they are more commonly benign.

(secondaries are due to direct spread from adjacent malignancies or metastatic diseases)

Most primary benign intracardiac tumors have characteristic appearance. On the contrary, most malignancies appear as an eroding mass without any borders separating it from the surrounding, also it had been suggested that the malignant mass, if pedunculated, the pedicel will be relatively wider than those of the benign tumors. The final distinct diagnosis will always be made on the basis of a biopsy.

Cardiac myxoma:

The myxoma is a benign tumor most commonly occurring in the left atrium and arising from the IAS at site of fossa ovalis, however it can originate from any other site. The most common characteristic feature is the pedicle with oscillating characteristic motion. Right atrial myxoma is shown in diagram 200 and LA myxoma is shown in diagram 201.

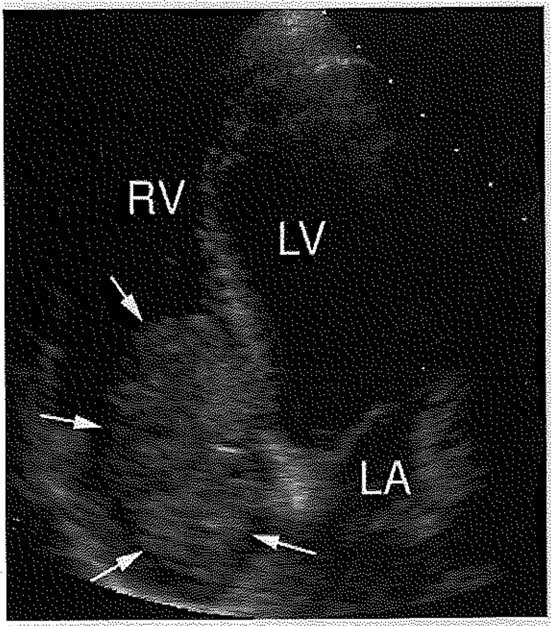


Diagram 200: a huge right atrial myxoma attached with a pedicle to the IAS.

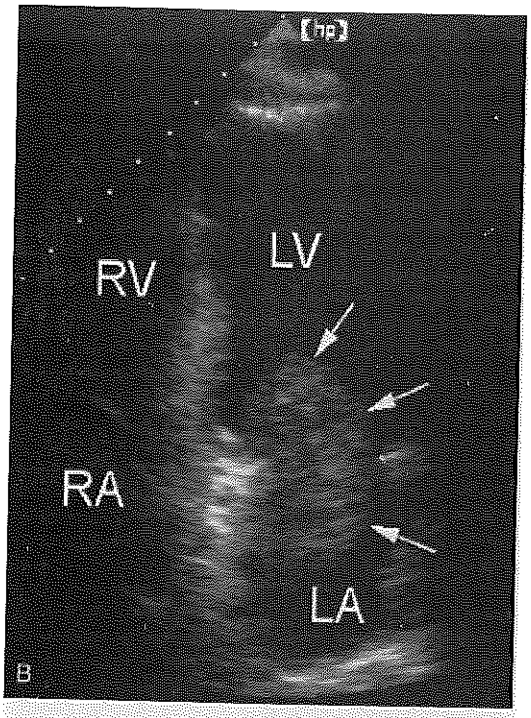


Diagram 201 : LA myxoma

N.B : Myxoma can send emboli.

Fibroelastoma :

This is another benign tumor of the heart most commonly arising from the tips of the intracardaic valves. Its highly mobile nature together with its position at the ventricular surface of the mitral valve (rather than at the upstream side of the valve), differentiate it from a vegetation. An example is shown in diagram 202. If the patient is suspected to suffer from infective endocarditis these fibroelastomas are commonly reported as vegetations.

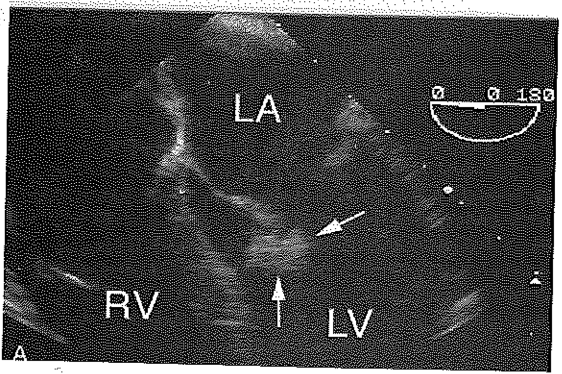


Diagram 202: A transoesophageal 4 chamber view, showing fibroelastoma of the mitral valve. Notice the attachment of the tumor to the ventricular surface of the mitral valve.

NB: lamble's excresences, which are considered by many authors as a normal variant can be easily confused for a fibroelastoma. See diagram 203.

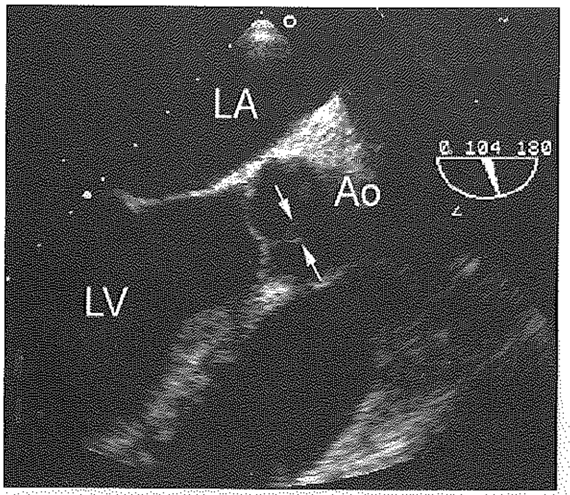


Diagram 203 : An example of Lambl's excrescence of the aortic valve is demonstrated (arrows).

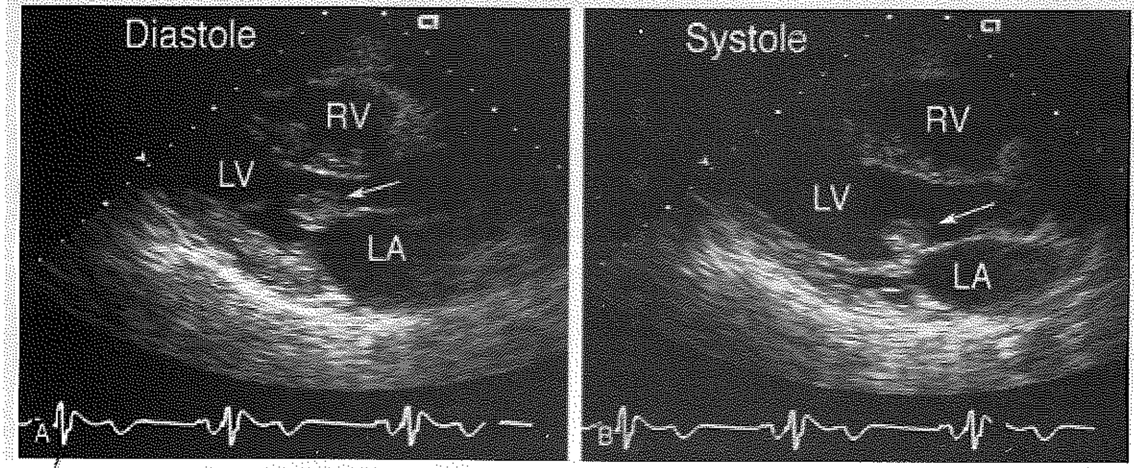


Diagram 204 : A blood cyst (arrow) within the anterior mitral leaflet is shown. The cyst is relatively immobile and the attachment is broad based. The mass is seen during diastole (A) and systole (B).

Examples of malignant intra cardiac tumors:

The definitive diagnosis will depend upon the biopsy if not contraindicated.

See diagrams 205 to 208.

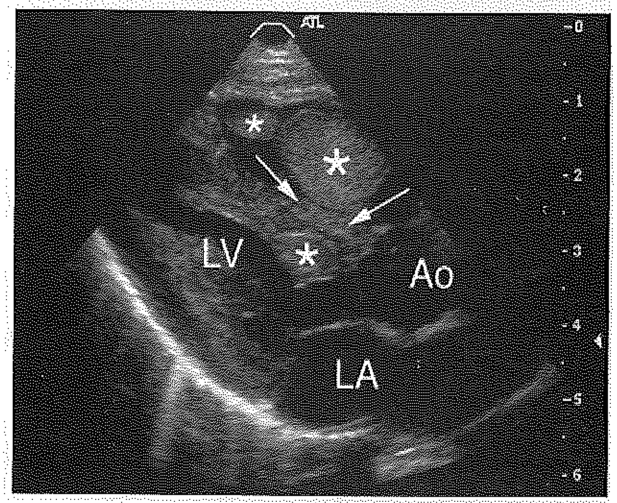


Diagram 205: Rhabdomyoma is a common pediatric tumor (as shown by asterisks and between the two arrows) .

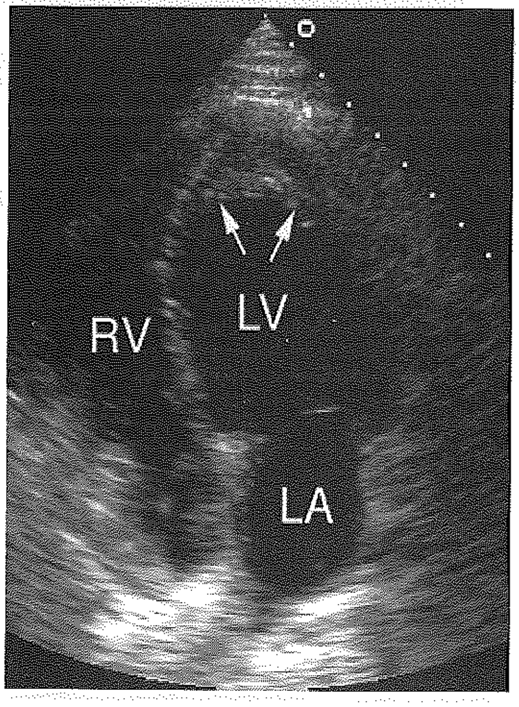


Diagram 206 : An example of endocardial fibroelastosis is shown

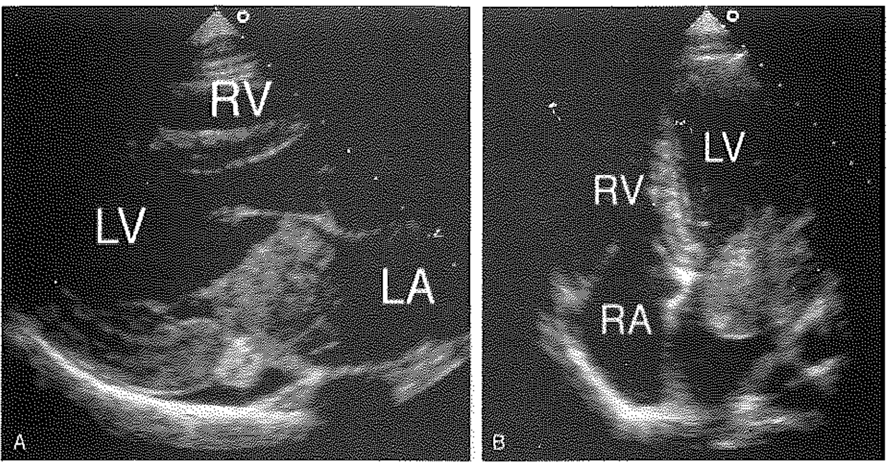


Diagram 207 : An example of angiosarcoma is provided. Notice the similarty between this tumor and the myxoma, however the absence of the pedicle excludes the myxoma as a diagnosis.

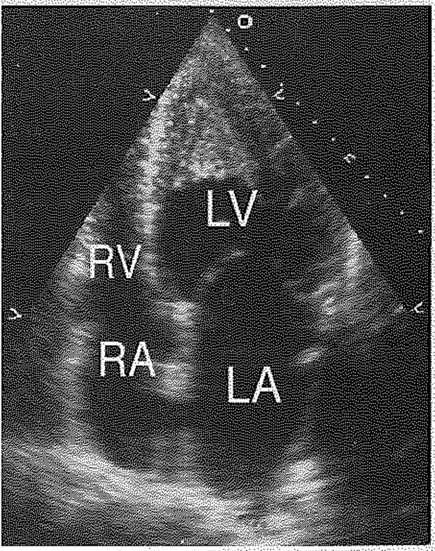


Diagram 208 : Endocardial thickening and fibrosis are characteristics of hypereosinophilic syndrome.

Summary of masses :

A mass is either normal or abnormal.

Abnormal masses are either thrombus/ vegetation or tumor.

Tumors are either primary(mostly benign) or malignant(mostly metastatic).

Chapter 19 : Artifacts in echocardiography

I. By 2D:

The echocardiography pictures acquisition depends on assuming several facts, once any fact assumed is not accomplished due to any factor, an artifact will occur.

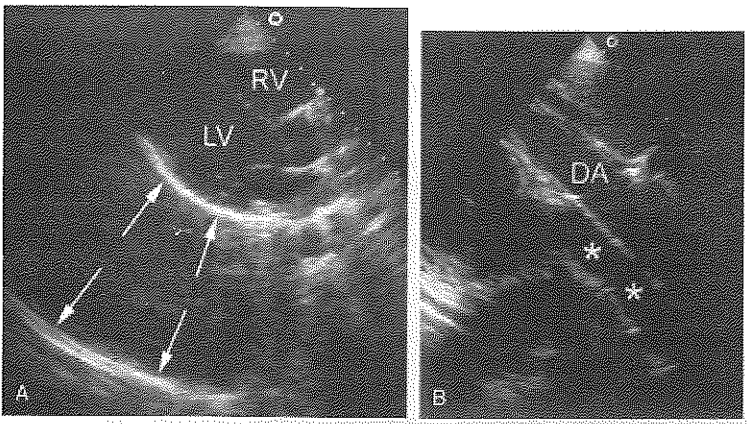
The impulses sent by the probe reflect back again only one time from the reflector (structure), so we only see one image.

a) Mirror Image:

What if the impulse actually reflected back from the structure to the probe and the chest wall, then bounced again from the chest wall to the structure then reflected back again to the probe?
The result is that, the probe will receive the echoes two times from the same structure after a doubled time. Remember from the "range equation" that we actually see distances that are originally calculated from time intervals, so a doubled time means a doubled distance. So the image will be created twice. One image is formed from the first reflection and another image will be formed at a doubled distance due to the second bounce.

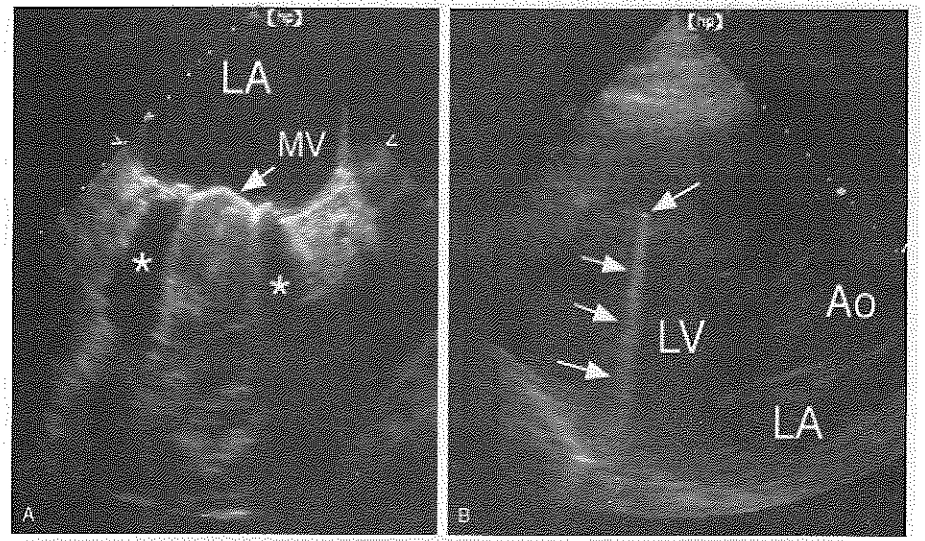
b) Reverberation Artifact

If the **bounce** is only one time, it will lead to mirror image. If many bounces, they will lead to many hyperechoic lines distal to the structure known as reverberation artifacts.
This phenomenon will occur in the presence of very strong reflectors such as prosthetic mechanical valves, where the echoes strongly bounce between the mechanical valve and the chest wall so many times.



Reverberation artifacts are demonstrated. A: The source of the artifact is the posterior pericardium, which is a very strong reflector. This creates the illusion of a second structure behind the heart. In this case, the second line of echoes (far arrows) is twice the distance from the transducer as the actual pericardial echoes. B: A second lumen appears just distal to the descending aorta (DA) in this subcostal view. The illusion of a second vessel was apparent with two-dimensional imaging (*, B)

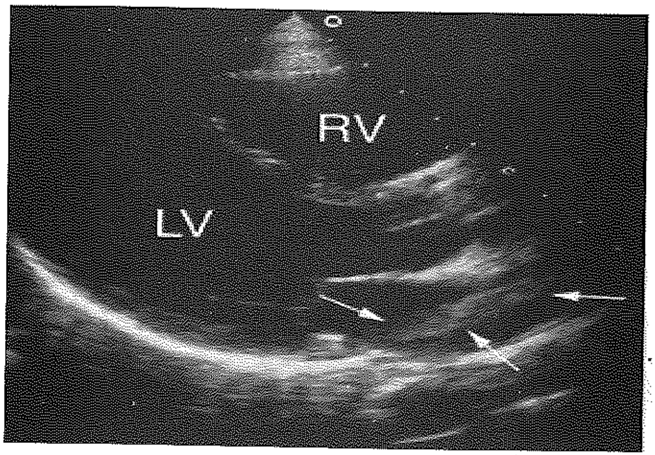
c) *Shadowing:*



The concept of shadowing is demonstrated and compared with reverberations. A: A St. Jude mitral prosthesis (MV) is present. The echo-free space beyond the sewing ring (*) represents shadowing behind the strong echo-reflecting sewing ring. The cascade of echoes directly beyond the prosthetic valve itself that extend into the left ventricle (LV) represent reverberations. B: A shotgun pellet within the heart (arrow) casts a series of reverberations into the left ventricle.

N.B in this diagram reverberation artifacts are also present shown as the white lines.

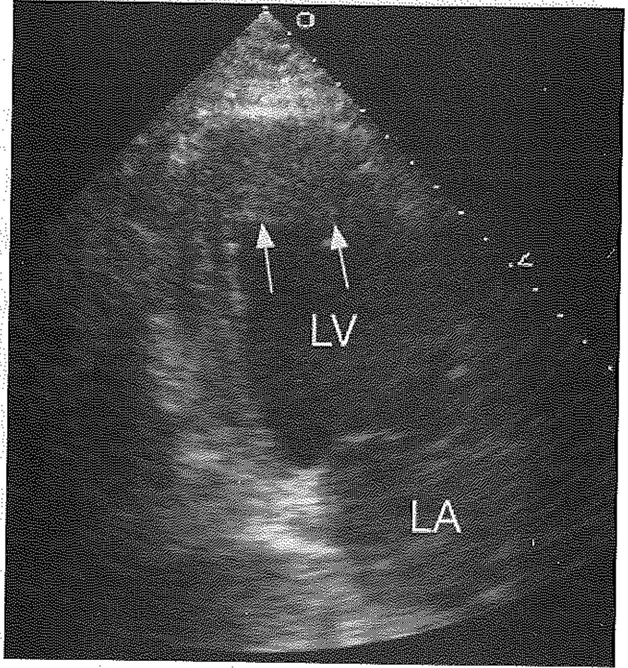
d) Side lobe artifacts:



The strong echoes produced by the posterior mitral anulus and atrioventricular groove produce a side lobe artifact that appears as a mass within the left atrium

Side lobes artifacts

e) Near field clutter:



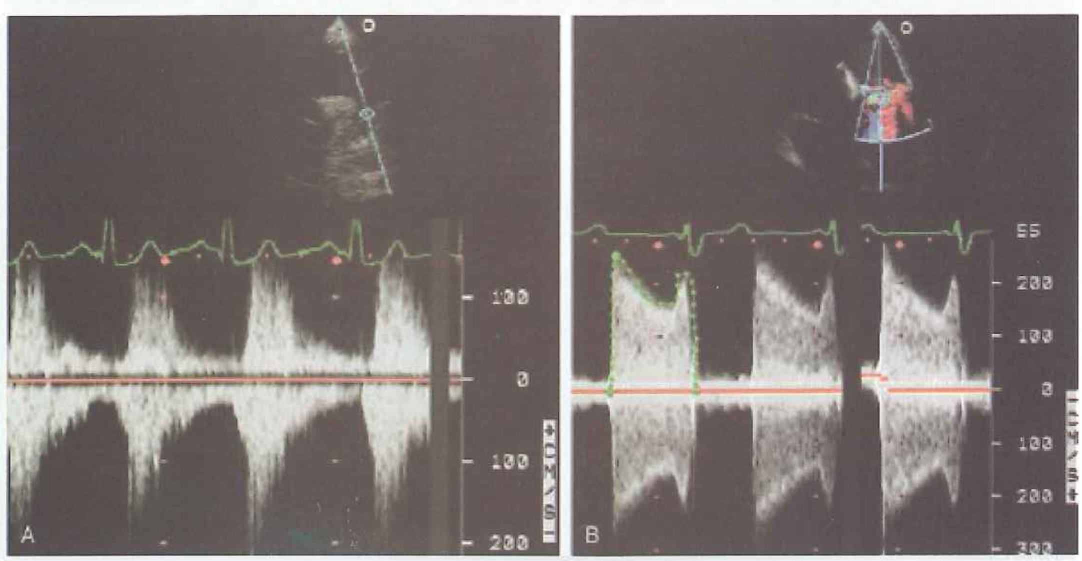
This apical two-chamber view demonstrates an artifact called near field clutter (arrows). This is the result of high-amplitude oscillations emitted by the transducer and is a common source of misinterpretation

Near field clutter

II. Doppler artifacts :

a) Aliasing : see before

b) Mirror image Doppler artifacts: due to very strong Doppler reflectance.



Two examples of mirror image artifact are demonstrated. A: descending aortic flow appears to occur both above and below the baseline. B: A stenotic porcine mitral valve is recorded with pulsed wave Doppler imaging. The intensity of the signal results in a classic mirror image artifact.

c) Artifacts in color Doppler due to high gain settings :

Too much gain can create a mosaic distribution of color signals throughout the image. (see before)

Too little gain eliminates all but the strongest Doppler signals and may lead to significant underestimation of jet area. (see before)

Low Nyquist limit will give similar effects as the high gain settings and vice versa.

d) Ghosting:

This is a phenomenon in which short bursts of color occur over parts of the picture. Ghosts are usually bleed into the tissue and are out of the clinical scenarios. This is due to the presence of strong reflectors such as prosthetic valves. They tend to be very transient and do not correspond to expected CW Doppler.

N.B: Ghosting is most problematic when color flow images are frozen to analyze or planimeter a jet.

Chapter 20: The echocardiography examination and reporting.

Take an oral consent in the presence of a nurse.

Complete chest exposure.

Subject all the cardiac structures to all the echocardiography techniques and maneuvers.

You can start either by the apical 4 ch view or the PLAX view.

In the **EMSE** we prefer to localize the apex first and start with the Ap 4 ch view.

- Bring a typical Ap 4 ch view :
  Examine all cardiac structures by 2 D imaging (LV, RV, IVS , LA, RA , IAS , MV, TV, tilt and look for the AV), align the color Doppler over the mitral and tricuspid valves then open the aorta with the color Doppler still playing, look for any mosaic patterns.
  examine the IVS for any defects.
  Align CW Doppler over MV, TV then tilt to examine the aortic valve by CW. If any high velocity jets are elicited they must be localized by the PW Doppler.
  Aligh PW Doppler over the tips of the MV, ask the patient to perform valsalva (if not contra indicated)
  Align M.mode over the lateral annuals of the tricuspid valve to measure TAPSE.
- Rotate the probe to image the Ap 2 ch view. Repeat the same steps for the miral valve.
- Rotate the probe to image the Ap 3 ch view. Repeat the same steps for the mitral and aortic valves .

- Bring a typical PLAX view. Examine the cardiac structures by 2 D imaging. Use the color Doppler over the mitral and aortic valves then examine the IVS for any defects.

- Align M.mode study first over the LV (at the tips of the MV to calculate EF), then over the aorta and LA. (measure the dimensions)
- Bring the RV inflow/ outflow (if needed)
- Rotate the probe for the short axis orientation

Tilt the probe to image the 3 levels : great vessels level, mitral valve level, and papillary muscle level.

In every view examine the heart by 2 D echo, M.mode and Doppler (align M.mode over the tips of the mitral valve to calculate the EF).

- Bring the SSLA view and align Doppler study over the ascending & descending aorta and the aortic arch.
- Bring the subcostal 4 ch view and reexamine the patient as before.
- Never tell the patient that the study is complete until you revise your report.

EMSE clinical tips for the beginner echocardiographer :

The dot of the probe is directed towards the left shoulder in all views except PLAX and it's equivalent Ap 3 chamber view where the dot is directed towards the right shoulder.

In the parasternal position all rotations will be in a clockwise manner.

In the apical position all the rotations will be in an anticlock wise manner.

Remember that the heart can have different axes and positions.

The one million dollars rule is : **SEARCH FOR THE APEX.**

Almost always use the **EMSE** step wise approach to find and optimize the view illustrated in chapter 1 "echo anatomy", never use haphazard random motions to find the view.

EMSE step wise approach to adjust the views "The summary" :

Use pure sliding motions to localize the heart (the only contracting organ in the chest !) always slide from lateral to medial with the U/S beam parallel to the long axis of the heart.

Always use the pendular "rocking motions" to centralize any view in the middle of the screen.

Once the view is centralized, do not add any further pendular motions.

- For the apical views :
    1) Tilting motions will optimize the LA and further titling motion will open the aortic valve.
    2) Rotational motion will optimize the RV.
    3) If the RV is too much centralized then you are already in a too much medial position, try a minimal lateral slide.
    4) If the RV is too much to the left of the screen and the LV is too much centralized in the middle of the screen, you are in a too much lateral position, so try a minimal medial slide.
- For the PLAX view :
    1) Tilting motion will optimize the LVOT and the aortic valve.
    2) Further tilting motions will image the RV inflow/ outflow view.
    3) If the LVOT is clear and the aortic valve is not optimized try minimal clockwise rotation.
    4) Rotational motions will adjust the U/S beam exactly over the long axis of the heart. i.e. will overcome a foreshortened LV .
    5) If there is marked variation with respiration ask the patient to shortly hold his breath, or to be more convenient try minimal upward/ down ward sliding.
- For the PSAX view:

    Rotate the probe in a clockwise manner until the LV becomes a complete circle, regardless the direction of the dot !

    Excessive rotation in the PSAX orientation will image the LV as an ovoid structure which will lead to an overestimation of the LVEDD.
- For the SSLA view:

    3 easy steps that must be followed in order :

    Place the probe in the supra sternal notch with dot directed towards the chin of the patient, then:
    1) Apply pendular motion to direct the U/S beam towards the heart.
    2) Apply fine rotational motion in a clockwise manner (try to keep the U/S beam directed towards the heart and not towards the patient's throat!!).
    3) Apply leftward tilting motion until the aortic arch is imaged.

N.B :

4) Never combine 2 types of motion together. Your first enemy is the falsely added on sliding motions.
5) Your second challenge is to prevent the probe from rotating while tilting and vice versa.

If you do not have full anatomical imagination and understanding of the anatomy of the cut section and the heart, leave the probe ! You will be wasting your time.

The main pitfalls that lead to failure to obtain the view are :

1) Failure to direct the U/S beam towards the heart.
2) Failure to align the U/S beam parallel to the desired imaging plane.
3) Aggressive hand motions and/or unsupported hand. (Always rest your hand over the patient, and handle the probe using your finger tips)

TTE echocardiography report

Various methods were suggested to write a complete and satisfactory TTE report.

Some suggested that the report should contain the 2 D findings and measurements then the M.mode measurements, then the Doppler calculations.

Others suggested that every cardiac structure should be satisfied separately in terms of all echocardiography maneuvers, i.e. 2 D echo. M.mode and Doppler.

Another way is to comment on the function and dimensions of every cardiac structure.

In bed side echo / emergency echo, only the positive findings are to be reported, with all the other normal findings just reported as "Normal".

In the **EMSE**, we are aware of the long echocardiography patient lists that face the Egyptian echocariographer. It was inevitable that we must organize our echocardiography reporting form that completely satisfies all the needed data the referring doctor will need and also confines the time to its minimal.

EMSE TTE report

Left ventricle : Dimensions → M.mode derived dimensions (3 diastolic measurements and the 3 systolic measurements of the IVS, LVEDD and PW)

Functions :
- → systolic : →by 2D echo → eye balling
  - →sympson's (if needed)
  - → By M.mode (M.mode derived EF)
  - → By Doppler (stroke volume)
- →diastolic →E/A ratio
  - → TDI
  - → Pulmonary venous flow (if needed)

Right ventricle : Dimensions → 2D derived dimensions from Apical 4 ch view.

Functions :
- → systolic : →by 2D echo → eye balling
  - →Simpson's (if needed)
  - → By M.mode → TAPSE
  - → By Doppler (stroke volume)
- →diastolic →E/A ratio

IVS : Intact or not ? If not, describe & report accordingly.

LA : Dimensions → M.mode derived dimensions in end-diastole (from the PLAX view)

Functions : Is the patient in sinus rhythm or not? (Other calculations are confined to research work & only done when they are thought to affect the clinical decision)

RA : Dimensions → 2 D echo derived dimensions in end-diastole in an Ap 4 ch view.

IAS : Intact or not ? (If abnormal, describe & report accordingly)

MV: MVA must be reported by both PHT method and planimetery.

Report peak velocity if normal, and mean PG only in stenotic valves.

The valve could either be → normal
- →stenotic→ valve area & MPG
  - Valve thickness and mobility by 2 D echo
- →regurgitates → report accordingly using the color Doppler.
  - →the shape of the valve by 2 D echo. (tethered/ poor cooptation)

TV : as mitral valve. (but the valve area is least important)

AV : peak velocity should be used with normal valves

PPG and MPG with abnormal valves.

Continuity equation should be used only if it is thought to affect the clinical decision.

The valve could either be → Normal
- →Stenotic→ valve area & MPG
  - Valve thickness and mobility by 2 D echo
- →Regurgitating → report accordingly using color Doppler.
  - →the shape of the valve by 2 D echo. (tethered/ or poor cooptation)

PV : as aortic valve (but the continuity equation is much less important)

The great vessels : aorta → M.mode measurement (PLAX view at the sinus of valsalva)

Main pulmonary artery, left and right pulmonary arteries : by 2 D imaging from PSAX view

Pulmonary artery pressure : EPASP = peak PG of the Tricuspid regurgitation + RA estimated pressure

Pericardium : →2 D echo : either normal, effusion, thickened.

→M.mode (only when needed or if 2 D echo not conclusive)

→ color Doppler (only when needed, as e.g. in a ruptured aneurysm; or if 2 D echo not conclusive)

Regional wall motion abnormalities: → 2 D echo

→M.mode . Either report as normal or only mention the abnormal segments.

Masses : should be thoroughly searched for.

Reported mainly by 2 D echo : size, site, shape, pedicle, echo density (see masses)

M.mode → (only when needed or if 2 D echo not conclusive)

Color Doppler →(only when needed or if 2 D echo not conclusive)

N.B : for every word and number written in the report you must document your proof in the form of a clear echo picture with a clear measure, printed or recorded on a CD.

Good luck with your echocardiography examination

Dr Mohamed Mamdouh Elsedafy
Registered Cardiac Sonographer, USA

Important numbers and equations in echocardiography

Velocity of Sound in Air and Various Types of Tissues

Medium	*Velocity (m/sec)*
Air	330
Fat	1450
Water	1480
Soft tissue	1540
Kidney	1560
Blood	1570
Muscle	1580
Bone	4080

Period of U/S wave : 0.1- 0.5 us

Pulse duration: 0.5-3 us

PRP: 0.1-1 ms

F= 1 MHz- 12 MHz

PRF= 1 KHz- 10 KHz

λ= 0.1-0.8 mm

spatial pulse length = 0.1-1mm

LARD =0.05-0.5mm

Half value layer thickness (penetration depth) = 0.25-1cm

Intensity = 0.001-100 W/cm^2

Impedance = 1.25-1.75 Mega Rayls

1 scan line of the color Doppler = 10 pulses of U/S

Mechanical index for the use of contrast echo = 1.3

Quality factor = Resonant frequency/ Band width , quality factor = number of the cycles in one pulse (2-4)

F= materials propagation speed (mm/us) / double the material's thickness

$dB = 10 \log P_2/P_1$

2014 AHA/ACC Valvular Heart Disease Guideline

Table 6. Stages of Valvular AS

Stage	Definition	Valve Anatomy	Valve Hemodynamics	Hemodynamic Consequences	Symptoms
A	At risk of AS	• Bicuspid aortic valve (or other congenital valve anomaly) • Aortic valve sclerosis	• Aortic V_{max} <2 m/s	• None	• None
B	Progressive AS	• Mild-to-moderate leaflet calcification of a bicuspid or trileaflet valve with some reduction in systolic motion or • Rheumatic valve changes with commissural fusion	• Mild AS: Aortic V_{max} 2.0–2.9 m/s or mean ΔP <20 mm Hg • Moderate AS: Aortic V_{max} 3.0–3.9 m/s or mean ΔP 20–39 mm Hg	• Early LV diastolic dysfunction may be present • Normal LVEF	• None
C: Asymptomatic severe AS					
C1	Asymptomatic severe AS	• Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening	• Aortic V_{max} ≥4 m/s or mean ΔP ≥40 mm Hg • AVA typically is ≤1.0 cm^2 (or AVAi ≤0.6 cm^2/m^2) • Very severe AS is an aortic V_{max} ≥5 m/s or mean ΔP ≥60 mm Hg	• LV diastolic dysfunction • Mild LV hypertrophy • Normal LVEF	• None: Exercise testing is reasonable to confirm symptom status
C2	Asymptomatic severe AS with LV dysfunction	• Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening	• Aortic V_{max} ≥4 m/s or mean ΔP ≥40 mm Hg • AVA typically ≤1.0 cm^2 (or AVAi ≤0.6 cm^2/m^2)	• LVEF <50%	• None
D: Symptomatic severe AS					
D1	Symptomatic severe high-gradient AS	• Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening	• Aortic V_{max} ≥4 m/s or mean ΔP ≥40 mm Hg • AVA typically ≤1.0 cm^2 (or AVAi ≤0.6 cm^2/m^2) but may be larger with mixed AS/AR	• LV diastolic dysfunction • LV hypertrophy • Pulmonary hypertension may be present	• Exertional dyspnea or decreased exercise tolerance • Exertional angina • Exertional syncope or presyncope
D2	Symptomatic severe low-flow/low-gradient AS with reduced LVEF	• Severe leaflet calcification with severely reduced leaflet motion	• AVA ≤1.0 cm^2 with resting aortic V_{max} <4 m/s or mean ΔP <40 mm Hg • Dobutamine stress echocardiography shows AVA ≤1.0 cm^2 with V_{max} ≥4 m/s at any flow rate	• LV diastolic dysfunction • LV hypertrophy • LVEF <50%	• HF • Angina • Syncope or presyncope
D3	Symptomatic severe low-gradient AS with normal LVEF or paradoxical low-flow severe AS	• Severe leaflet calcification with severely reduced leaflet motion	• AVA ≤1.0 cm^2 with aortic V_{max} <4 m/s or mean ΔP <40 mm Hg • Indexed AVA ≤0.6 cm^2/m^2 and • Stroke volume index <35 mL/m^2 • Measured when patient is normotensive (systolic BP <140 mm Hg)	• Increased LV relative wall thickness • Small LV chamber with low stroke volume • Restrictive diastolic filling • LVEF ≥50%	• HF • Angina • Syncope or presyncope

AR indicates aortic regurgitation; AS, aortic stenosis; AVA, aortic valve area; AVAi, aortic valve area indexed to body surface area; BP, blood pressure; HF, heart failure; LV, left ventricular; LVEF, left ventricular ejection fraction; ΔP, pressure gradient; and V_{max}, maximum aortic velocity.

Table 9. Stages of Chronic AR

Stage	Definition	Valve Anatomy	Valve Hemodynamics	Hemodynamic Consequences	Symptoms
A	At risk of AR	• Bicuspid aortic valve (or other congenital valve anomaly) • Aortic valve sclerosis • Diseases of the aortic sinuses or ascending aorta • History of rheumatic fever or known rheumatic heart disease • IE	• AR severity: none or trace	• None	• None
B	Progressive AR	• Mild-to-moderate calcification of a trileaflet valve bicuspid aortic valve (or other congenital valve anomaly) • Dilated aortic sinuses • Rheumatic valve changes • Previous IE	• **Mild AR:** ○ Jet width <25% of LVOT; ○ Vena contracta <0.3 cm; ○ RVol <30 mL/beat; ○ RF <30%; ○ ERO <0.10 cm^2; ○ Angiography grade 1+ • **Moderate AR:** ○ Jet width 25%–64% of LVOT; ○ Vena contracta 0.3–0.6 cm; ○ RVol 30–59 mL/beat; ○ RF 30%–49%; ○ ERO 0.10–0.29 cm^2; ○ Angiography grade 2+	• Normal LV systolic function • Normal LV volume or mild LV dilation	• None
C	Asymptomatic severe AR	• Calcific aortic valve disease • Bicuspid valve (or other congenital abnormality) • Dilated aortic sinuses or ascending aorta • Rheumatic valve changes • IE with abnormal leaflet closure or perforation	• Severe AR: ○ Jet width ≥65% of LVOT; ○ Vena contracta >0.6 cm; ○ Holodiastolic flow reversal in the proximal abdominal aorta ○ RVol ≥60 mL/beat; ○ RF ≥50%; ○ ERO ≥0.3 cm^2; ○ Angiography grade 3+ to 4+; ○ In addition, diagnosis of chronic severe AR requires evidence of LV dilation	C1: Normal LVEF (≥50%) and mild-to-moderate LV dilation (LVESD ≤50 mm) C2: Abnormal LV systolic function with depressed LVEF (<50%) or severe LV dilatation (LVESD >50 mm or indexed LVESD >25 mm/m^2)	• None; exercise testing is reasonable to confirm symptom status
D	Symptomatic severe AR	• Calcific valve disease • Bicuspid valve (or other congenital abnormality) • Dilated aortic sinuses or ascending aorta • Rheumatic valve changes • Previous IE with abnormal leaflet closure or perforation	• Severe AR: ○ Doppler jet width ≥65% of LVOT; ○ Vena contracta >0.6 cm, ○ Holodiastolic flow reversal in the proximal abdominal aorta, ○ RVol ≥60 mL/beat; ○ RF ≥50%; ○ ERO ≥0.3 cm^2; ○ Angiography grade 3+ to 4+; ○ In addition, diagnosis of chronic severe AR requires evidence of LV dilation	• Symptomatic severe AR may occur with normal systolic function (LVEF ≥50%), mild-to-moderate LV dysfunction (LVEF 40% to 50%), or severe LV dysfunction (LVEF <40%); • Moderate-to-severe LV dilation is present.	• Exertional dyspnea or angina or more severe HF symptoms

AR indicates aortic regurgitation; ERO, effective regurgitant orifice; HF, heart failure; IE, infective endocarditis; LV, left ventricular; LVEF, left ventricular ejection fraction; LVESD, left ventricular end-systolic dimension; LVOT, left ventricular outflow tract; RF, regurgitant fraction; and RVol, regurgitant volume.

Table 11. Stages of MS

Stage	Definition	Valve Anatomy	Valve Hemodynamics	Hemodynamic Consequences	Symptoms
A	At risk of MS	• Mild valve doming during diastole	Normal transmitral flow velocity	None	None
B	Progressive MS	• Rheumatic valve changes with commissural fusion and diastolic doming of the mitral valve leaflets • Planimetered MVA >1.5 cm^2	• Increased transmitral flow velocities • MVA >1.5 cm^2 • Diastolic pressure half-time <150 ms	• Mild-to-moderate LA enlargement • Normal pulmonary pressure at rest	None
C	Asymptomatic severe MS	• Rheumatic valve changes with commissural fusion and diastolic doming of the mitral valve leaflets • Planimetered MVA ≤1.5 cm^2 • (MVA ≤1.0 cm^2 with very severe MS)	• MVA ≤1.5 cm^2 • (MVA ≤1.0 cm^2 with very severe MS) • Diastolic pressure half-time ≥150 ms • (Diastolic pressure half-time ≥220 ms with very severe MS)	• Severe LA enlargement • Elevated PASP >30 mm Hg	None
D	Symptomatic severe MS	• Rheumatic valve changes with commissural fusion and diastolic doming of the mitral valve leaflets • Planimetered MVA ≤1.5 cm^2	• MVA ≤1.5 cm^2 • (MVA ≤1.0 cm^2 with very severe MS) • Diastolic pressure half-time ≥150 ms • (Diastolic pressure half-time ≥220 ms with very severe MS)	• Severe LA enlargement • Elevated PASP >30 mm Hg	• Decreased exercise tolerance • Exertional dyspnea

The transmitral mean pressure gradient should be obtained to further determine the hemodynamic effect of the MS and is usually >5 mm Hg to 10 mm Hg in severe MS; however, due to the variability of the mean pressure gradient with heart rate and forward flow, it has not been included in the criteria for severity.

LA indicates left atrial; LV, left ventricular; MS, mitral stenosis; MVA, mitral valve area; and PASP, pulmonary artery systolic pressure.

Table 13. Stages of *Primary* MR

Grade	Definition	Valve Anatomy	Valve Hemodynamics*	Hemodynamic Consequences	Symptoms
A	At risk of MR	• Mild mitral valve prolapse with normal coaptation • Mild valve thickening and leaflet restriction	• No MR jet or small central jet area <20% LA on Doppler • Small vena contracta <0.3 cm	• None	• None
B	Progressive MR	• Severe mitral valve prolapse with normal coaptation • Rheumatic valve changes with leaflet restriction and loss of central coaptation • Prior IE	• Central jet MR 20%–40% LA or late systolic eccentric jet MR • Vena contracta <0.7 cm • Regurgitant volume <60 mL • Regurgitant fraction <50% • ERO <0.40 cm^2 • Angiographic grade 1–2+	• Mild LA enlargement • No LV enlargement • Normal pulmonary pressure	• None
C	Asymptomatic severe MR	• Severe mitral valve prolapse with loss of coaptation or flail leaflet • Rheumatic valve changes with leaflet restriction and loss of central coaptation • Prior IE • Thickening of leaflets with radiation heart disease	• Central jet MR >40% LA or holosystolic eccentric jet MR • Vena contracta ≥0.7 cm • Regurgitant volume ≥60 mL • Regurgitant fraction ≥50% • ERO ≥0.40 cm^2 • Angiographic grade 3–4+	• Moderate or severe LA enlargement • LV enlargement • Pulmonary hypertension may be present at rest or with exercise • C1: LVEF >60% and LVESD <40 mm • C2: LVEF ≤60% and LVESD ≥40 mm	• None
D	Symptomatic severe MR	• Severe mitral valve prolapse with loss of coaptation or flail leaflet • Rheumatic valve changes with leaflet restriction and loss of central coaptation • Prior IE • Thickening of leaflets with radiation heart disease	• Central jet MR >40% LA or holosystolic eccentric jet MR • Vena contracta ≥0.7 cm • Regurgitant volume ≥60 mL • Regurgitant fraction ≥50% • ERO ≥0.40 cm^2 • Angiographic grade 3–4+	• Moderate or severe LA enlargement • LV enlargement • Pulmonary hypertension present	• Decreased exercise tolerance • Exertional dyspnea

*Several valve hemodynamic criteria are provided for assessment of MR severity, but not all criteria for each category will be present in each patient. Categorization of MR severity as mild, moderate, or severe depends on data quality and integration of these parameters in conjunction with other clinical evidence.

ERO indicates effective regurgitant orifice; IE, infective endocarditis; LA, left atrium/atrial; LV, left ventricular; LVEF, left ventricular ejection fraction; LVESD; left ventricular end-systolic dimension; and MR, mitral regurgitation

Table 14. Stages of *Secondary* MR

Grade	Definition	Valve Anatomy	Valve Hemodynamics*	Associated Cardiac Findings	Symptoms
A	At risk of MR	• Normal valve leaflets, chords, and annulus in a patient with coronary disease or cardiomyopathy	• No MR jet or small central jet area <20% LA on Doppler • Small vena contracta <0.30 cm	• Normal or mildly dilated LV size with fixed (infarction) or inducible (ischemia) regional wall motion abnormalities • Primary myocardial disease with LV dilation and systolic dysfunction	• Symptoms due to coronary ischemia or HF may be present that respond to revascularization and appropriate medical therapy
B	Progressive MR	• Regional wall motion abnormalities with mild tethering of mitral leaflet • Annular dilation with mild loss of central coaptation of the mitral leaflets	• ERO <0.20 cm^2† • Regurgitant volume <30 mL • Regurgitant fraction <50%	• Regional wall motion abnormalities with reduced LV systolic function • LV dilation and systolic dysfunction due to primary myocardial disease	• Symptoms due to coronary ischemia or HF may be present that respond to revascularization and appropriate medical therapy
C	Asymptomatic severe MR	• Regional wall motion abnormalities and/or LV dilation with severe tethering of mitral leaflet • Annular dilation with severe loss of central coaptation of the mitral leaflets	• ERO ≥0.20 cm^2 † • Regurgitant volume ≥30 mL • Regurgitant fraction ≥50%	• Regional wall motion abnormalities with reduced LV systolic function • LV dilation and systolic dysfunction due to primary myocardial disease	• Symptoms due to coronary ischemia or HF may be present that respond to revascularization and appropriate medical therapy
D	Symptomatic severe MR	• Regional wall motion abnormalities and/or LV dilation with severe tethering of mitral leaflet • Annular dilation with severe loss of central coaptation of the mitral leaflets	• ERO ≥0.20 cm^2† • Regurgitant volume ≥30 mL • Regurgitant fraction ≥50%	• Regional wall motion abnormalities with reduced LV systolic function • LV dilation and systolic dysfunction due to primary myocardial disease	• HF symptoms due to MR persist even after revascularization and optimization of medical therapy • Decreased exercise tolerance • Exertional dyspnea

*Several valve hemodynamic criteria are provided for assessment of MR severity, but not all criteria for each category will be present in each patient. Categorization of MR severity as mild, moderate, or severe depends on data quality and integration of these parameters in conjunction with other clinical evidence.

†The measurement of the proximal isovelocity surface area by 2D TTE in patients with secondary MR underestimates the true ERO due to the crescentic shape of the proximal convergence.

2D indicates 2-dimensional; ERO, effective regurgitant orifice; HF, heart failure; LA, left atrium; LV, left ventricular; MR, mitral regurgitation; and TTE, transthoracic echocardiogram.

Table 17. Stages of TR

Stage	Definition	Valve Anatomy	Valve Hemodynamics*	Hemodynamic Consequences	Symptoms
A	At risk of TR	**Primary** • Mild rheumatic change • Mild prolapse • Other (e.g., IE with vegetation, early carcinoid deposition, radiation) • Intra-annular RV pacemaker or ICD lead • Postcardiac transplant (biopsy related) **Functional** • Normal • Early annular dilation	• No or trace TR	• None	• None or in relation to other left heart or pulmonary/pulmonary vascular disease
B	Progressive TR	**Primary** • Progressive leaflet deterioration/destruction • Moderate-to-severe prolapse, limited chordal rupture **Functional** • Early annular dilation • Moderate leaflet tethering	**Mild TR** • Central jet area <5.0 cm^2 • Vena contracta width not defined • CW jet density and contour: soft and parabolic • Hepatic vein flow: systolic dominance **Moderate TR** • Central jet area 5–10 cm^2 • Vena contracta width not defined but <0.70 cm • CW jet density and contour: dense, variable contour • Hepatic vein flow: systolic blunting	**Mild TR** • RV/RA/IVC size normal **Moderate TR** • No RV enlargement • No or mild RA enlargement • No or mild IVC enlargement with normal respirophasic variation • Normal RA pressure	• None or in relation to other left heart or pulmonary/pulmonary vascular disease
C	Asymptomatic, severe TR	**Primary** • Flail or grossly distorted leaflets **Functional** • Severe annular dilation (>40 mm or 21 mm/m^2) • Marked leaflet tethering	• Central jet area >10.0 cm^2 • Vena contracta width >0.7 cm • CW jet density and contour: dense, triangular with early peak • Hepatic vein flow: systolic reversal	• RV/RA/IVC dilated with decreased IVC respirophasic variation • Elevated RA pressure with "c-V" wave • Diastolic interventricular septal flattening may be present	• None, or in relation to other left heart or pulmonary/pulmonary vascular disease
D	Symptomatic severe TR	**Primary** • Flail or grossly distorted leaflets **Functional** • Severe annular dilation (>40 mm or >21 mm/m^2) • Marked leaflet tethering	• Central jet area >10.0 cm^2 • Vena contracta width >0.70 cm • CW jet density and contour: dense, triangular with early peak • Hepatic vein flow: systolic reversal	• RV/RA/IVC dilated with decreased IVC respirophasic variation • Elevated RA pressure with "c-V" wave • Diastolic interventricular septal flattening • Reduced RV systolic function in late phase	• Fatigue, palpitations, dyspnea, abdominal bloating, anorexia, edema

*Several valve hemodynamic criteria are provided for assessment of severity of TR, but not all criteria for each category will necessarily be present in every patient. Categorization of severity of TR as mild, moderate, or severe also depends on image quality and integration of these parameters with clinical findings.

CW indicates continuous wave; ICD, implantable cardioverter-defibrillator; IE, infective endocarditis; IVC, inferior vena cava; RA, right atrium; RV, right ventricle; and TR tricuspid regurgitation.

Table 18. Stages of Severe TS

Stage	Definition	Valve Anatomy	Valve Hemodynamics	Hemodynamic Consequences	Symptoms
C, D	Severe TS	- Thickened, distorted, calcified leaflets	- T ½ ≥190 ms - Valve area ≤1.0 cm^2	- RA/IVC enlargement	- None or variable and dependent on severity of associated valve disease and degree of

Table 19. Stages of Severe Pulmonic Regurgitation

Stage	Definition	Valve Anatomy	Valve Hemodynamics	Hemodynamic Consequences	Symptoms
C, D	Severe PR	- Distorted or absent leaflets, annular dilation	- Color jet fills RVOT - CW jet density and contour: dense laminar flow with steep deceleration slope; may terminate abruptly	- Paradoxical septal motion (volume overload pattern) - RV enlargement	- None or variable and dependent on cause of PR and RV function

CW indicates continuous wave; PR, pulmonic regurgitation; RV, right ventricular; and RVOT, right ventricular outflow tract. (247)

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Stage	Definition	Valve Anatomy	Valve Hemodynamics	Hemodynamic Consequences	Symptoms
C, D	Severe PS	- Thickened, distorted, possibly calcified leaflets with systolic doming and/or reduced excursion - Other anatomic abnormalities may be present, such as narrowed RVOT	- V_{max} >4 m/s; peak instantaneous gradient >64 mm Hg	- RVH - Possible RV, RA enlargement - Poststenotic enlargement of main PA	- None or variable and dependent on severity of obstruction

PA indicates pulmonary artery; PS, pulmonic stenosis; RA, right atrium; RV, right ventricle; RVH, right ventricular hypertrophy; RVOT, right ventricular outflow; and V_{max}, maximal pulmonic valve jet velocity. (9)